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ON CONGENITAL HEART AFFECTIONS, ESPECIALLY IN
RELATION TO THE DIAGNOSIS OF THE VARIOUS
MALFORMATIONS.

THE WIGHTMAN LECTURE FOR 1909.*

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GENTLEMEN,—In acknowledging the honour that you have conferred upon me by inviting me to deliver the Wightman Lecture, I must confess that the acceptance of this distinction proved far easier than the selection of a suitable subject for the occasion.

In choosing congenital heart affections, I have been guided by the fact that these disorders are essentially and peculiarly infantile and childish complaints, and that it affords me an opportunity of passing in review the work done in this direction by the late Society for the Study of Disease in Children, and of its successor the Section for the Study of Disease in Children of the Royal Society of Medicine.

Passing now to the subject of congenital affections of the heart, I will first draw your attention to the known causes which bring about the various departures from the normal, which are classed under the heading "Congenital Heart Affections."

* Delivered at the Section for the Study of Disease in Children of the Royal Society of Medicine, June the 24th, 1909.

The coincidence of congenital malformations of the heart with malformations in other regions of the body is a matter of everyday experience. Heart malformations are not always accompanied by other congenital bodily defects, but they are sometimes found in association with such diverse structural faults as hare-lip, cleft palate, ill-developed teeth, supernumerary auricles, and supernumerary nipples, polydactylism, syndactylism, webbed fingers, steeple-skull, defects in the abdominal wall, herniæ, ill-developed and absent rib cartilages, undescended testis, atresia of the anus and rectum, post-anal dimple, congenital opacity of the cornea, coloboma iris, congenital defects of the orbits, congenital ptosis, not to mention many other congenital irregularities of development which are obvious to the naked eye—cretinism, for instance—which sometimes bear them company.

Of internal mal-developments which are not obvious on inspection there are numerous illustrations, such as unilateral kidney, horse-shoe kidney, double ureters, absent spleen, supernumerary spleens, malpositions of the intestines, atrophic patches in the choroid, transposition of the viscera, four lobes to the lungs, and an accessory bronchus amongst other anomalies.

Deaf-mutism is sometimes an associated condition, and the coincidence of Mongolian imbecility with congenital heart malformations has been illustrated by cases and morbid specimens, which have been shown from time to time at the meetings of the late Society for the Study of Disease in Children, and also at those of its successor, the Section for the Study of Disease in Children of the Royal Society of Medicine. Those adverse influences which lead to the production of congenital defects of the nervous system are also apt to be associated with malformations of the heart, and illustrating this feature I have at the present time under my care a boy of ten years with Friedreich's disease, in whom the two conditions are combined.

That congenital malformations of the heart are sometimes hereditary has been proved by several observers—Ferrannini, De la Camp, Orth, and others—who have recorded examples of this as a family complaint. Among my own series I have illustrations of two cases in the children of one family, and of three cases in the children of the other.* Among the latter, one, an example of steeple skull,

* "Two Sisters showing Malformations of the Skull and other Congenital Abnormalities," 'Reports of the Society for the Study of Disease in Children,' vol. i, pp. 110-118, illustrated. "Case of Acrocephaly with other Congenital Malformations," 'Proc. Roy. Soc. Med.,' Section for the Study of Disease in Children, vol. ii, No. 2, pp. 45-53. Ditto, "Autopsy," vol. ii, No. 7, pp. 199, 200, illustrated. Preparations of the Skull and Brain: Nos. in Catalogue, 360'50, 360'51, 360'52, Royal College of Surgeons Museum, Lincoln's Inn Fields.

came to autopsy, and the heart, like the child, was fantastic, and could not be placed in any usual anatomical grouping. The foramen ovale was widely open and divided into two by a bridge of tissue. Behind this was another hole in the septum, and above this hole the septum was minutely fenestrated. The right auricular appendix was very large and muscular, $1\frac{1}{4}$ in. long by $\frac{3}{4}$ in. broad at its broadest part, and it and its corresponding auricle made an elongated chamber about 2 in. long and a little more than $\frac{3}{4}$ in. broad behind. The tricuspid orifice was small, $\frac{3}{16}$ in. only, with natural though small valves, and passed into the cavity of the right ventricle, which was so small that it would only admit a pea. The pulmonary artery was smaller than the aorta, and was situated directly over the ventricular cavity. The thickness of the ventricular wall was $\frac{3}{16}$ in., and at its upper and outer part was a distinct muscular hump, on which the enlarged auricular appendix rested. The left auricle was more muscular than natural, and the left ventricle was enormously hypertrophied, its walls measuring $\frac{7}{16}$ in. thick, which so encroached on its cavity that its capacity appeared to be even less than that of the right. The mitral orifice, which was larger than the tricuspid, was $\frac{5}{16}$ in. across, and its valves were natural. The aorta, which had a right-angled bend on it like that of a ventilating shaft on a steamer, was thick walled; its valves were normal. The greater part of the heart was made up of the left ventricle and the right auricle and appendix. There was no bruit during life.

Since heart malformations have been shown to be a family complaint in some instances, it must be admitted that inherited as well as acquired defects in the sperm and germ-cells of the parents are of some importance in the production of malformations of the heart, as in other regions, for erratic tendencies to produce deformities when once acquired are handed down from parents to children.

Inquiry has been directed to the state of the mother's health, both bodily and mental, during the pregnancy to serve as an explanation for the production of congenital cardiac defects. Maternal impressions and trivial injuries have been, and are still frequently, advanced to explain away congenital anomalies of the heart and elsewhere, because mothers have been brought up in such beliefs, and readily recall all sorts of strange sights and slight mishaps incident to the pregnancy, if it be the misfortune of the offspring to present any abnormalities of mind or body. When it is realised that the heart is perfectly formed, though in miniature, in seven weeks' time from the date of conception, the various examples of congenital heart disease

that have been recorded by J. Lewis Smith and others, following upon frights and so on during the last two or three months of pregnancy, these psychical disturbances cannot possibly have had any effect upon the foetal heart. Maternal impressions must, I think, be thrown into the waste-heap of other ill-founded superstitions.

But it is otherwise when the bodily health of the mother becomes the subject of scrutiny. In regard to definite illnesses affecting the mother during the pregnancy, it is surprising how few mothers give a history of suffering any illness during that period. In 100 examples of my own of congenital malformation of the heart, in a large number the parents and their families were apparently free from rheumatic taint, as no history of that complaint was admitted. In a few instances the parents had suffered from rheumatic fever, but in no case was there any evidence in this series of maternal rheumatic fever taking place during pregnancy. Therefore the commonest cause for endocarditis in children was conspicuous by its inconspicuousness in my pre-natal cases. But Theodore Fisher* narrates that in a case of foetal endocarditis three months before the birth of the infant the mother suffered from severe pain in the left knee, and for several days she could only get about the house with difficulty. This fact is of importance, because it shows that rheumatic fever in the mother is not essential to the production of foetal endocarditis, and it suggests that those painful bodily ailments in the mother during the carrying, which are grouped under the generic term "rheumatic," may be the cause of foetal endocarditis in the infant.

Endocarditis in the foetus has been attributed to typhoid fever, influenza, pneumonia, and other maternal infections. But these diseases arising during the pregnancy are infrequent in comparison with the number of cases of foetal endocarditis for which a ready explanation is not forthcoming. It would appear that the placenta is able to be penetrated and the foetus infected by micro-organisms and toxins, quite apart from maternal indisposition. Perhaps favouring the toxin origin is the fact that the form of the endocarditis is frequently of the sclerotic or chronic variety. I have found tolerably frequently among my cases a history of miscarriages, premature births, or decease within a short interval of birth. Such misfortunes are generally regarded as strong presumptive evidence of syphilis. I have, however, discovered no reason to ascribe many of these accidents of the pregnancies to syphilis, and it is more than

* "Congenital Aortic Stenosis from a Child, aged 4 months," 'Reports of the Society for the Study of Disease in Children,' vol. ii, p. 16.

likely that they were due to anatomical abnormalities in the foetus which were incompatible with its life.

Syphilis, of course, is naturally uppermost in the mind as an explanation for congenital affections of the heart, but it does not appear to be a very important factor in the creation of cardiac abnormalities. The number of congenital syphilitics that I have seen with congenital heart malformations has been very few; and the number of my cases of congenital heart disease that could be ascribed to syphilis was trifling—only three. Bearing upon this question, it may be noted that Hochsinger found congenital heart disease only seven times in 500 cases of congenital syphilis. The statement has been made at our various meetings, and has not been warmly controverted, that congenital syphilis is of considerable importance in the production of congenital affections of the heart; but this view does not appear to be warranted by my own clinical experience. There is bacteriological evidence to prove that malformations of the cardiac valves may be associated with spirochæte infection. Landouzy and Laederich* found the mitral and the tricuspid valves puckered and thickened, and partly adherent to the ventricular septum in an obviously syphilitic infant of two and a half months. The right side of the heart was hypertrophied and the aorta small. Spirochætes were found in the papules of the skin and in the supra-renal glands, but not elsewhere. It would appear likely that the virus of syphilis exerts a two-fold influence, viz. by the action of the poison on the endocardium, with the production of foetal endocarditis, and by its action as a producer of malformations without endocarditis.

There is strong presumptive evidence that alcohol has a decided influence in the production of deformities, and clinical experiences are borne out by experimental observations on the lower animals. In Hodge's experiments, where alcohol was administered to two parent dogs, of twenty-four puppies born many were deformed or dead. Féré, also, in controlled experiments on chicken's eggs exposed to the vapour of alcohol, found that a number of the chicks were found dead or stillborn, and displayed various deformities.

That immoderate drug-taking may be a cause for congenital abnormalities, I have an example in the person of a lady who was a confirmed morphino-maniac at the conception and during the pregnancy, and with the result that an imbecile child was born to her.

Tuberculosis in the parents has been credited with the production of hypoplasia of the aorta.

* 'La Presse Medicale,' May, 1907, No. 43, p. 337.

Several examples of foetal endocarditis have been shown to the Society for the Study of Disease in Children, or have been spoken of at its meetings. Theodore Fisher's case* of aortic disease occurred in a child, aged 4 months. The flaps of the semi-lunar valves were much thickened and adherent. Some thickening of the flaps of the mitral valve and its chordæ tendineæ was also present. G. W. Nash, of Bedford, at the same meeting, narrated a case of a child who died, aged 6 months, from mitral stenosis. The mitral valve was puckered and diminished. This he attributed to sepsis from defective drainage, as the mother developed pneumonia from that cause after the confinement. In a case reported by the late A. E. Sansom† in a baby, aged 2 months, there was a ring of granulations of endocarditis, and the mitral valve was thickened. The changes were of the rheumatic character, but the mother, so he stated, had never suffered from rheumatism, and he went on to say that endocarditis in intra-uterine life was so rare that it was desirable that every case should be reported. At a later meeting during the same session, Fisher‡ showed the heart of an infant, aged 4 months, in which the aortic and pulmonary valves displayed large organised vegetations. There was an intra-pericardial septal defect between the pulmonary artery and aorta, which extended downwards into the upper part of the septum ventriculorum. The pulmonary artery had two valves, and the cardiac defects were associated with somatic defects in the shape of a hare-lip and the rectum and vagina sharing a common aperture. Fisher attributed the endocarditis to defective house-drains during the pregnancy. In Ayrolle's case§ there was marked stenosis of the mitral valve, on which appeared numerous endocarditic vegetations. The infant died ten days after its birth. In a case recorded by Cotton|| granulations were present both on the mitral and on the cusps of the aortic valve.

Planchu and Gardère¶ have recently recorded a case of endocarditis of the pulmonary artery in an infant, aged 6 weeks. The artery was bivalved, the lower surface of the cusps being rugose and showing numerous vegetations and ulcerations. The cardiac septa

* *Loc. cit.*

† "The Pathological Anatomy and the Mode of Development of Mitral Stenosis in Children," 'Medical Society's Transactions,' vol. xiii, p. 143.

‡ "A Specimen of Congenital Heart Disease, showing Vegetations on the Pulmonary and Aortic Valves," 'Reports of the Society for the Study of Disease in Children,' vol. ii, p. 155.

§ 'Rev. Mens. des Mal. de l'Enf.,' 1885.

|| 'Trans. American Pediatric Society,' vol. xii, 1900, p. 142.

¶ 'Arch. de Med. des Enf.,' tome xii, pp. 201-208, March, 1909.

were so defective that the organ was practically one of two chambers. The great vessels were transposed and the pulmonary artery was very narrow, and would only admit a small sound. There were associated visceral defects. In the heart of an infant, aged 25 days, recently exhibited by André Moussous* the mitral valve showed signs of recent endocarditis. The orifice of the pulmonary artery was obliterated by a membranous arched diaphragm produced by the coalescence of the sigmoid valves. Above the tricuspid in the cavity of the auricle there was a pre-orificial narrowing in the shape of a rigid ring hardly admitting a goose-quill. The foramen ovale valve had a fenestrated appearance.

Fœtal heart murmurs have been recorded by various writers. Tricuspid regurgitation has been detected *in utero* by Peter,† who heard a systolic bruit replacing the first sounds of the heart. The child was stillborn. The tricuspid valve was covered with vegetations and tied down by shortened chordæ tendineæ. Hochsinger‡ quotes two cases, those of Barth and Hennig. In the former the child was also stillborn and there was endocarditis of the tricuspid valve. In Hennig's case a double murmur was due to endocarditis of the aortic valves. In Christopher's case, reported to the American Pediatric Society, the aortic and tricuspid valves showed verrucose thickening, and the pulmonary artery took origin from the aorta. In Wetherill's case§ the murmur persisted after birth and was attributed to stenosis of the pulmonary artery, while in Hall's case|| the bruit disappeared and was ascribed to patent ductus arteriosus, which became obliterated.

In the production therefore of congenital affections of the heart two distinct processes can be seen in action, viz. developmental disturbance and inflammation. Both can operate without the other and both are frequently combined. Those mal-influences, inherited and acquired, which preside over the sperm and germ-cells have been passed in review, as also the causes of fœtal endocarditis. The results of fœtal endocarditis are seen in the shape of sclerotic valves and on these valves it is not uncommon to find recent endocarditic vegetations. It also frequently happens that in association with congenital developmental anomalies the mitral and tricuspid valves

* 'Société de Méd. S. de Chir. de Bordeaux in Gaz. Hebd. des Sc. Médic.,' March the 8th, 1908. 'Archives des Maladies du Cœur,' vol. i, 1908.

† 'Congenital Affections of the Heart,' p. 72, 1894.

‡ 'Die Auscultations des Kindlichen Herzens.'

§ 'Amer. Journ. of Obstetrics,' January, 1904, p. 36.

|| 'Archives of Pediatrics,' 1897, p. 905.

are found to be somewhat thickened and crumpled at the edges though perfectly efficient, suggesting that they have undergone a mild endocarditis during foetal life. It is in stenosis of the pulmonary artery that valvulitis and anomaly are so frequently combined. The points at issue are these: Are the patent septum ventriculorum and the atrophied pulmonary artery the result of increased blood-pressure in the right ventricle and starvation of the artery—that is, purely mechanical, or are the defects in the artery and the septum ventriculorum of developmental origin, and the endocarditis of the pulmonary valve an indication of its increased liability to attack by reason of developmental anomaly?

As Osler has pointed out, it does not seem reasonable to suppose that inflammation could be confined to such tiny structures as the pulmonary valves of a foetus less than two months old. In a small number of cases endocarditis occurs without septal defect, and in these the inflammation must have taken place at a much later date after the completion of the septum. Many developmental cardiac anomalies occur without valvular inflammation similar to the cases in which inflammation sometimes participates. It is obvious, therefore, that inflammation is not necessary. It would seem in some of these cases that endocarditis merely fastens upon a weak spot the weakness inherent to mal-development because it is frequently seen that malformed as well as once diseased valves are prone to attack.

It is only in a very small proportion of the cases of congenital defect that endocarditis, when it is limited to the valves, can be viewed as the cause of the disease, and such arise after the heart has been completed. The bulk of the cases of congenital malformation of the heart appear to be due to developmental errors when seen by the light of modern research on cardiac development.

Morbus Cœruleus.

Morbus cœruleus and congenital malformations of the heart have been looked upon as synonyms, but without reason, for children with severe congenital morbus cordis are frequently not blue, even when excited or crying. Many verified cases of congenital morbus cordis have been recorded where cyanosis was not a symptom during life. On the other hand, chronic lung* disease may be associated with extreme and chronic cyanosis, a characteristic example of which I exhibited to the Society for the Study of Disease in Children in 1906.

* "A Case of Cirrhosis of the Lung and Bronchiectasis," 'Reports of the Society for the Study of Disease in Children,' vol. vi, p. 59.

Some children with congenital morbus cordis are fresh-coloured. Others are strikingly pale. In some the lips are dark crimson, perhaps inclined to blueness rather than redness, or to turn lilac on crying. Or the complexion may be quite good and only the fingers dusky and cold-looking. The majority, however, are cyanosed more or less, and all gradations of cyanosis may be noticed, from that of a dark rubicund face with similar coloured ears and reddened finger- and toe-tips, or a state of rather dusky-blue lips and cheeks and tongue and a similar condition of the extremities, to one where the face is plum-coloured, the conjunctivæ suffused, the buccal mucous membrane and tongue the colour of a slate, and the body generally dusky. In the majority of those who are blue the cyanosis dates from birth, but in others the children show no signs of blueness until after an attack of one of the exanthems, or following bronchitis or bronchopneumonia, with or without whooping-cough.

Increase of cyanosis in those already blue and of paroxysmal onset is sometimes a symptom. In one cyanosed infant of mine, aged 2 months, who developed paroxysmal cyanosis, it was associated with a loud systolic bruit audible all over the front and back of the chest, and most marked at the second *right* cartilage. In another infant, aged 1 month, the only lesion that I could find after death was a patent foramen ovale. During the attacks that I witnessed in these infants the pulse, which was at first rapid, became gradually and quickly slower, and the breathing, which had been accelerated, ceased, and the infant remained for several minutes in a state of complete apnoea, during which time the cyanosis became extreme in the face and lips and tongue, and on the body and limbs. The conjunctivæ were insensitive and the pupils slightly contracted. If left undisturbed after some four or five minutes the infant gasped, the breathing recommenced, the colour returned and the eyes were opened, only to be followed in about half an hour by a recurrence of the same symptoms. Attacks such as these suggest to my mind a central origin, possibly of the medulla, for they bear some resemblance to attacks of unexpected cyanosis and apnoea which sometimes take place during the course of tuberculous meningitis, and with a suddenly fatal result. Such patients can certainly be temporarily revived, and perhaps on several occasions, if their paroxysms of cyanosis are treated by artificial respiration. Morrow* has described recurrent cyanosis associated with very shallow respiration in two infants. One child, at the time of the report, was living and

* "Recurrent Attacks of Cyanosis in Infants," 'Montreal Med. Journ.,' February, 1905.

well. The other, a premature infant, died of asthenia, but there was no post-mortem. Morrow quotes Holt as having described similar symptoms in infants in whom atelectasis was found at autopsy.

Leconte* has reported two examples of paroxysmal cyanosis in children of three and a half and four and a half years old, which appeared for the first time in the first case a month previous and in the second child at two years of age. The attacks were sudden, severe and frequently repeated, and came on without exciting causes. The children became suddenly cyanosed, limp and semi-conscious, respiration was noisy and difficult and the paroxysms lasted two or three minutes. One child had a loud systolic bruit at the second left interspace, and the other transposition of the viscera with a loud systolic murmur at the second right interspace.

Variot, under the term "congenital intermittent cyanosis," has drawn attention to the onset of cyanosis under exciting conditions, energetic movements and forced expiration. In some of these children I have observed attacks of palpitation at the same time. I have seen† cyanosis disappear in an infant of five months and be followed by occasional attacks of cyanosis without any apparent exciting causes. In a boy of four years under my care, whose face and cheeks were like those of a ruddy man of forty, but in whom there was no polycythæmia, though undoubted congenital heart disease, there were frequent paroxysms of cyanosis.

Sudden increase of cyanosis in some of these cyanotic children is very apt to be followed by convulsions. During the attacks the pulse becomes barely perceptible at the wrist, and death is very likely to take place at these occasions.

Cyanosis becomes aggravated when dilatation of the right ventricle takes place, and the veins of the body become overcharged with blood. Microscopical examination of the organs‡ shows extreme capillary dilatation, with thickening and a tendency to fibrillation, as also venous engorgement and thickening in the brain, kidney, liver, heart, and elsewhere. I examined many microscopic sections of the skin of the patient, some twenty years ago, from whom this description of the capillaries is taken, but I found no capillary dila-

* 'La Clin. Infant.,' April, 1907, No. 8, p. 235.

† "A Case of Congenital Morbus Cordis in a Mongol," 'Reports of the Society for the Study of Disease in Children,' vol. viii, p. 278.

‡ "Microscopical Changes in the Organs found in a Case of Cyanosis with Congenital Malformation of the Heart by the Lecturer," 'St. Thomas's Hospital Reports,' vol. xviii; also "Congenital Affections of the Heart," pp. 25, 26, 27.

tations there. Hochsinger* states that the skin capillaries are dilated and tortuous in these cases, especially in the peripheral parts of the body. So wide and so tortuous were the vessels in the case I examined, that while, for instance, in the kidney the afferent arteries of the Malpighian corpuscles admitted but two or three red corpuscles abreast, the enormously dilated afferent veins contained seven or eight or more. Loutaud states that the veins are not only dilated, but have hypertrophied muscular walls. The infrequency of general œdema in congenital cyanosis in children has been ascribed to this hypertrophy. But this absence of œdema occurs in acquired heart disease in children, and is not peculiar to congenital heart disease, and I have also met with several examples of œdema in the last stages of congenital heart disease in spite of these structural alterations in the veins and capillaries.

By far the most common malformations of the heart in association with cyanosis are those cases where the pulmonary artery or the conus are either absent, rudimentary or constricted. Next in frequency in the list of malformations, though only to about one seventh of their number, is transposition of the pulmonary artery and aorta. Apart from these mal-developments, which form the bulk of congenital cardiac anomalies, the malformations which give rise to cyanosis are numerous and various.

Various views have been and are still held as to the causation of cyanosis, the criticism that I have to make about them being that it is sought to explain all cases of cyanosis by one theory, whereas the cause is often complex and differs in different cases.

The oldest theory, that of venous stasis, is that of Morgagni, which has numerous adherents. Another view is that cyanosis is due to the admixture of venous and arterial blood, an explanation which has been supported by Meckel and others. Alexander Morison,† who critically examined some seventy-five cases of congenital heart disease, came to the conclusion that "the main, though not the only factor in the production of cyanosis is the inadequate aid afforded to the circulation by diminished lung functions."

In regard to the second theory, the admixture of venous and arterial bloods, the mingling of these bloods may be most free, and yet no cyanosis be produced. In Breschet's famous case the left arm was supplied by the pulmonary artery, the corresponding subclavian artery arising from it, but the limb was not cyanosed.

* *Loc. cit.*, p. 463.

† 'Cyclopædia of Diseases of Children' (Keating), vol. ii, part ii, p. 762.

Further lung functions may also be considerably encroached upon in a case where the mingling of blood is free to take place and yet no cyanosis appears.* In a child, aged 9 months, under my care, the right and left auricles practically formed one large chamber, and a patent ductus arteriosus in great measure supplied the descending aorta, yet the addition of extensive pneumonic consolidation of the lung was not sufficient to curtail its oxygenating functions, for the infant *was at no time cyanosed*. The parts of the lungs still in action were perfectly healthy.

In another infant of mine, aged 5 months, whose case I have not published before, with transposition of the pulmonary artery and aorta, and a large patent ductus arteriosus, there were the following additional anomalies: A single auricle, with two appendices and a rudimentary membranous septum. There was no trace of a tricuspid valve. The right ventricle was rudimentary, and there was a perforate septum ventriculorum, the size of a crow-quill, leading into it and situated underneath the pulmonary valves. The lungs were quite healthy and pink. The lungs sometimes show an appreciable amount of atelectasis in some infants suffering from congenital malformation of the heart and without the production of cyanosis.

In many cases the explanation is invited, not that the lungs cannot oxygenate properly, but that they are prevented from dealing with a sufficient proportion of the total quantity of venous blood throughout the body by reason of various defects in the pulmonary region, perhaps combined with weakness of the heart muscle.

Cyanosis, therefore, appears to depend in some cases not upon defective aëration from defects in the lungs, but to be due to the conformation of the heart, which by reason of faulty construction and physical weakness is placed at a mechanical disadvantage, and is not adapted to deliver a sufficient quantity of venous blood to the lungs to be aërated.

But this will not explain all cases, for in some the lungs are not healthy and display considerable microscopical alterations. So wide and so tortuous were the thickened lung capillaries in the case of atresia of the pulmonary artery I examined that those in the alveoli admitted six or more red corpuscles abreast. Changes such as these, permeating as they do the whole of the lung structure, are

* "Defective Auricular Septum; Pulmonary Artery larger than Aorta, the Thoracic Aorta being practically a continuation of that Vessel by Ductus Arteriosus; Isthmus Aortæ stenosed," 'Reports of the Society for the Study of Disease in Children,' vol. viii, p. 231.

not conducive to proper aëration. It would appear, then, that diffuse and not localised structural alterations of the lung tissues are favourable to the production of cyanosis. This condition of the lungs alone appears to be sufficient to explain the occurrence of cyanosis in bronchiectasis and chronic emphysema. In the former there is great thickening and dilatation of the alveolar capillaries, and in both diseases there is considerable destruction of them.

Here, then, are two factors in operation; the difficulty of getting the blood to the lungs to be aërated, and the difficulty of aërating the blood when it arrives there should it so happen that the lungs are not in a position to undertake the operation by reason of their structural defects.

Systemic venous congestion complicates matters; it does not produce them, for it is possible to have a considerable amount of cyanosis without venous congestion. When venous congestion takes place additional factors come into play which increase the cyanosis, and these are: (*a*) the formation of enlarged veins and tortuous capillaries throughout the body, and (*b*) increased viscosity of the blood leading to further retardation of its flow.

Cyanosis, I take it, is due to deficient oxygenation from whatever cause arising, and not necessarily to impaired lung functions.

The explanation for the tardy onset of cyanosis and its origin in sequence to a diffuse inflammation of the lungs, such as bronchopneumonia may probably be found in some cases in wide-spread alterations in the alveolar capillaries. What is required is that attention shall be paid to the lungs of all children dying from congenital morbus cordis. I have examined them microscopically in a few cases but with negative findings.

In other children perhaps the initial breakdown is in the muscle of the right side of the heart.

Clubbing of the Fingers and Toes and Osteo-arthritis.

Clubbing of the fingers and toes in congenital heart disease, the so-called Hippocratic fingers, depends almost entirely on congestive swelling and thickening of the soft parts of the terminal phalanges. The connective tissue is increased, and the capillaries are enlarged and increased in number.

The finger-tips are broadened and the nails cyanosed, a different appearance to that shown in chronic osteo-arthritis, where the nail is domed like a watch glass, curls over the finger-tip, and bears a striking resemblance to a parrot's beak, and is combined with

osteo-periostitis of the terminal phalanx. The finger looks like a drumstick, and the nail is pink coloured.

There appears to be two varieties of clubbed fingers; in one there is osteo-periostitis of the terminal phalanges, but in the other this has not yet been demonstrated. Whether there are really two varieties of clubbed fingers or whether the conditions are different stages of the same process has not yet been settled.

Sternberg* regards the Hippocratic type of clubbed fingers as the commencement of the process, and this is Walter's† opinion, who thinks the two conditions are closely allied. He also points out that in the majority of cases of osteo-arthropathy defective blood aëration has been a feature of the case.

Bamberger's and Marie's views are that the bone changes are due to the absorption of poisons generated at the seat of the disease, wherever that may be situated, and it has been shown that about one quarter of the cases have arisen from a variety of affections other than those of the lungs. But these views omit to take into consideration the influence of congestion in the production of the enlargement of the soft parts, which certainly is an important cause in congenital heart malformation, and may be contributory in chronic lung disease.

The toxic effects, however, of the cyanosis in congenital heart disease cannot be overlooked, because clubbing is sometimes seen in these cases without much evidence of congestion.

Béclère‡ looks upon clubbing and osteo-arthropathy as due to congestion and toxæmia. Both clubbing and osteo-periostitis may be confined to one limb, and such conditions have been brought about by the pressure of an aortic aneurysm obstructing its circulation. The explanation advanced for the clubbing and osteo-periostitis is that toxic substances generated at the extremity of the affected limb are retained in the tissues by reason of the venous obstruction.

The youngest child among my cases in which clubbing was obvious was aged $2\frac{1}{2}$ years. It was also stated to be present in one aged 7 months, in whom cyanosis was reported to be slight, but the physical signs were in favour of pulmonary stenosis.

* "Vegetationsstörungen und Systemerkrankungen der Knochen," 'H. V. Nothnagel's Spec. Pathol. u. Therapie Wien,' 1903, Bd. vii, Theil ii, p. 72.

† 'St. Thomas's Hospital Reports,' vol. xxiv, p. 1, 1895; *ibid.*, p. 105; 'Brit. Med. Journ.,' 1896, vol. i, p. 329, February the 8th.

‡ 'Semaine Médicale, 1901,' p. 94. 'Bulletins et Memoires de la Société Médicale des Hôpitaux,' 1901, vol. xviii, p. 283. 'Gazette des Hôpitaux,' 1904, p. 754.

Common as is this clubbing of the fingers and toes in congenital heart disease, nevertheless it is a feature of this disease which very often is wanting. It is in pulmonary atresia and stenosis that clubbing most commonly arises, and it is in these conditions that congestion is most frequently observed.

Osteo-arthritis of the Marie and Bamberger type, as osteo-periostitis of the distal extremities of the long bones as the seat of election with thickening of the carpal and tarsal bones and bulbous enlargement of the terminal phalangeal bones associated with parrot-beak-like nail changes and fingers of drumstick-like appearance is most rare in children. The number of cases reported can be counted on the fingers.

Those cases that have been recorded by Royal Whitman, Davis and Gillet have been associated with chronic lung diseases. Bamberger's case was aged 7 years, and suffered from pulmonary stenosis, congenital cyanosis, and a tuberculous lung. Thorburn's case had mitral disease only. A few cases in association with chronic lung disease have been recorded by Moussous, Gillet, Morzard, Marfan and Miller, in which the characteristic beak-like finger-nails occurring with osteo-periostitis of the terminal phalanges were the sole changes. D. F. M. Miller* provides a picture and a radiogram of a child, aged 8 years, and contrasts pictures of the fingers in the case with that of a case of congenital heart disease in a child, aged 7 years.

Of all the cases recorded that of Royal Whitman,† in a girl, aged 8 years, was the most extensive. There was a deposit of new periosteal bone upon the shafts of the phalanges, upon the metacarpal and metatarsal bones, and upon the lower third of the bones of the lower arm and leg. The knees and ankles and wrists were enlarged from thickening of the capsules and the soft parts and effusion into the joints. Under the section dealing with the blood changes I have drawn attention to a case of my own in a child aged $3\frac{9}{12}$ years with cirrhosis of the lung and bronchiectasis where the phalanges and metacarpal bones were thickened.

Cases have been recorded by Weill and Mouriquand‡ and by Variot in congenital heart disease in which to the naked eye the appearances of the finger-tips and toes, the wrists and the insteps

* 'Trans. Amer. Pediat. Soc.,' vol. xvi, pp. 267-77, 1904-05.

† "Secondary Pulmonary Hypertrophic Osteo-arthritis complicating Pott's Disease in a Child," 'Pediatrics,' vol. vii, 1899, pp. 154-162. Illustrated with photographs and a skiagram of the hands and forearms.

‡ 'Lyon Med.,' 1908, No. 15, p. 844.

at once suggested pulmonary osteo-arthropathy, but when radiograms were made it was found that only the soft parts were involved.

A case of osteo-arthropathy occurring in congenital heart disease in a woman, aged 21 years, has been recorded by Batty Shaw and R. Higham Cooper.* Clubbing of the fingers and toes and cyanosis were well marked, and there was a systolic murmur best heard over the third left interspace. There was no bony increase in the terminal phalanges of the fingers. The lower ends of the tibiæ and fibulæ were thickened. The bones of the forearms and wrists were slightly swollen, and also the shafts of the metacarpal and phalangeal bones. The opsonic index for tubercle was normal.

Ophthalmoscopic Appearances.

Ophthalmoscopic examination in cases of marked congestion reveals tortuous retinal blood-vessels of which the veins are very large. The blood in the arteries looks decidedly venous, though they are not quite so dark coloured as the veins. The red reflex, which is of cyanosed appearance, often appears to start from the physiological pit, and the number of vessels on the face of the disc appears to be greatly increased. In children where the cyanosis amounts to but little more than a ruddy bucolic appearance a want of the usual colour contrast between the arteries and veins is quite noticeable. The retinal blood-vessels early display evidences of congestion by enlargement and tortuosity of both arteries and veins. If there be considerable cyanosis retinæ, as sometimes is the case, without the associated appearance of curly retinal vessels, such an ophthalmoscopic finding, although not proof that there is no narrowing of the pulmonary region, nevertheless suggests that the pulmonary tract is either not involved or that right-sided muscular compensation is sufficient to overcome the obstruction.

Blood Changes.

Blood changes such as polycythæmia, macrocythæmia, and increased viscosity along with a by no means invariable increase in the hæmoglobin are not uncommon. In one blue child under my care, aged $2\frac{1}{2}$ years, with clubbed fingers and toes,† who had been blue since birth, there was a blood-count of 7,880,000 per c.mm., and the hæmoglobin was 122 per cent. That is the largest number of red cells that I have met with in congenital heart disease. The heart was

* 'Clin. Soc. Trans.,' vol. xl, p. 257, March the 22nd, 1907.

† 'Reports of the Society for the Study of Disease in Children,' vol. v, p. 48.

large, the interventricular and auricular septa defective, and the aorta rose from both ventricles. Hypoplasia of the pulmonary artery and its branches was a marked feature starting at the orifice.

In Planchu and Gardère's* infant, a child, aged 6 weeks, in whom cyanosis was slight but worse on crying, the polycythæmia amounted to 9,000,000 per c.mm. This was with a heart of practically two cavities. Bach,† in a child, aged 7 weeks, found no less than 11,400,000 per c.mm. There was atresia of the pulmonary artery, a patent ductus arteriosus, cicatricial occlusion of the tricuspid valve, and a rudimentary right ventricle.

Many of the children under my care with polycythæmia have shown a deficiency in the hæmoglobin rather than an increase. In a blue infant, aged 1 year, with clubbed fingers and toes and lips, and finger- and toe-tips of purple hue, the blood-count amounted to 5,400,000 per c.mm. red, with a hæmoglobin percentage of 55 only. In an infant with intermittent cyanosis Variot found intermittent polycythæmia. Weil‡ has shown in two children with morbus cœruleus and congenital morbus cordis increase in the red bone-marrow, and his observations have been confirmed. This increase of red blood-corpuscle producing areas must be viewed as compensatory to the state of chronic suffocation, which is the normal condition of these patients. Parkes Weber,§ when recently reporting a case of congenital heart disease with polycythæmia, has called attention to the observations by Lorrain Smith and H. L. M'Kisack on a boy, aged 12 years, with chronic cyanosis, who demonstrated not only a relative polycythæmia, but also that the total amount of blood in the body was far beyond the normal standard—nearly double. In Weber's case, a youth, aged 22 years, compensatory polycythæmia was extreme, the blood-count amounting to 10,300,000 per c.mm.

The most intense polycythæmia that I have met with in a child occurred in a case|| of cirrhosis and bronchiectasis of the lung in a boy, aged $3\frac{9}{12}$ years. He was very blue, clubbing of the fingers and toes was a marked feature, and the phalanges of the hands and the metacarpal bones appeared to be thickened.¶ There was extension of the cardiac dulness to the right. A blood-count gave 8,270,000 red corpuscles per c.mm.

* *Loc. cit.*

† 'Arch. f. Kinderheilk.,' p. 31, 1909.

‡ 'Compt. Rend. Soc. de Biol., Paris,' 1901, vol. liii, p. 713.

§ "A Case of Congenital Heart Disease," 'Edin. Med. Journ.,' 1909.

|| "A Case of Cirrhosis of the Lung, Bronchiectasis," 'Reports of the Society for the Study of Disease in Children,' vol. vi, p. 59.

¶ *Vide* observations on osteoarthropathy.

Among other symptoms of congenital morbus cordis respiratory acceleration is the rule, though it does not always occur when the child is at rest. In some cardiac pain is a feature. In a case reported by Hand junior,* in a case of pulmonary atresia, attacks of dyspnœa were associated with heart pain, an angina pectoris, and during the attacks the respirations were slow and laboured. The pulse is often accelerated. In only one form of malformation is it slowed, and that happens sometimes in pulmonary stenosis. The temperature is frequently subnormal. Growth and nutrition may both be faulty, and infantilism sometimes results.

Congenital Heart-murmurs.

Congenital heart-murmurs are of various characters. They may be heard as harsh, roaring, churning, growling, sawing, twanging, rushing, or rasping noises. Musical bruits are not common in my experience. If murmurs of such character be heard in a child under three years of age, and if they be conducted over wide areas, the presumption is strong that the affection is of congenital origin. Soft, low, very faint bruits are sometimes present. Murmurs are usually systolic, occasionally pre-systolic or diastolic, and there is that peculiar rumbling murmur carried through the systole and diastole, which points to a patent ductus arteriosus or a communication between the pulmonary artery and the aorta. Sometimes the bruits become inaudible shortly before death, or during a paroxysm of cyanosis, and I have noticed a loud murmur to almost disappear under the influence of an anæsthetic. A murmur may be detected at one auscultation, and cannot be heard at the next, and the causes for this temporary disappearance are not quite clear. Murmurs are also influenced by posture and by respiration. In regard to conduction, in some instances systolic bruits may be traced along the arteries over the brachials as far as the bends of the elbows, and also be heard in the thighs along the course of the femorals. They can not only be followed up the arteries of the neck, but exceptionally can be heard by applying the ear to the vertex.

It is now fifteen years or more since I first tried to arrive at some idea as to the nature of the malformation† by studying the murmurs produced at the various cardiac orifices. Since then I have been able to extend my clinical experiences, and the result of my observations, many of which have been verified, I will bring to your notice

* 'Trans. American Pediatric Society,' vol. xx, 1909, p. 10.

† 'Congenital Affections of the Heart; Stethoscopic Signs,' 1894.

under the various sections into which I have divided the subject now before us.

In only a small proportion of the cases are murmurs absent. In my series this occurred in 7 per cent.

Thrills are not infrequent; they are usually systolic, occasionally diastolic, or even pre-systolic. These purring tremors may be localised to the site at which they are produced or be of wide conduction. Further, the site of greatest intensity by no means necessarily corresponds to the seat of production. A systolic thrill of greatest intensity at the second left interspace and conducted towards the corresponding clavicle is, however, pathognomonic of patent ductus arteriosus. A systolic thrill may be felt at the episternal notch over the aorta and be conducted into the carotidis and subclavians.

Defects in the Interventricular Septum.

Defects in the interventricular septum are the most common of all cardiac malformations. They are associated mostly with stenosis and atresia of the pulmonary artery, transposition of the great vessels, and occlusion of the tricuspid orifice. The combination of pulmonary stenosis, deviation of the aorta to the right, and patent septum ventriculorum is one of the most usual forms of congenital morbus cordis. While interventricular septal defects are very common in association with other cardiac abnormalities they are rare without combined congenital cardiac defect. Starck* stated that he had never seen a defective septum without an associated anomaly of the pulmonary valves during life, but he had the record of one autopsy in which this rare condition existed. In a case recorded by Willcocks† in an infant, aged 3 weeks, there was a patent septum ventriculorum without other congenital defect. In a male under my care, aged 3 months, whose case has not been published, there was a perforation of this septum the size of a goose-quill just below the aortic valves. The sigmoid flexure was much enlarged and lay in the right iliac fossa. In a girl, aged 8 months, also hitherto unpublished, I found a septal perforation in the same situation. The perforation admitted a rod 5 mm. in diameter and the ductus arteriosus was patent to the extent of a rod of 6 mm. In a specimen from a child, aged 2 years,‡ I exhibited to the Society for the Study

* 'Pediatrics,' vol. ix, 1900.

† 'Path. Soc. Trans.,' 1886-7.

‡ 'Reports of the Society for the Study of Disease in Children, vol. vi, pp. 241-2.

of Disease in Children in 1906, there was a patent septum ventriculorum $\frac{1}{4}$ in. in diameter below the aortic valves. There were two cusps to the pulmonary valve and an ill-marked division in one of them, and the pulmonary artery was dilated. In a girl, aged 8 years, who died of malignant endocarditis of the aortic and mitral valves, this septal defect was the sole congenital anomaly, and it was not suspected during life. This defect may occur in association with irregularity in the distribution of the veins to the heart, such as in a case recorded by W. C. Chaffey,* of Brighton, which happened in a female infant, aged 10 weeks. A part of the blood returning from the head and upper extremity entered the heart by way of the left duct of Cuvier and the coronary sinus.

As was found in the cases I have just related, the commonest situation for perforate septum ventriculorum is directly beneath the aortic valves and just in front of the undefended space. In front of it lies the fleshy part of the septum and behind it the membranous. On the right side of the heart this opening will be found beneath the septal and anterior cusps of the tricuspid valve.

It is rare for a perforation to be found in the anterior fleshy part. In a case reported by Coupland † there was an aperture the size of a No. 12 catheter passing into the conus of the pulmonary artery just below the pulmonary valve. When viewed from the conus side an aortic cusp partially occluded the septal deficiency. Defects in the pars membranacea, the undefended space, are not uncommon, and perforations may be found there varying in size from a pin puncture to an area of considerable extent, bounded in front and below by the rounded muscular border of the ventricular septum. This latter was well seen in the heart of a Mongol, aged 5 months, with a single auricle, associated with fused mitral and tricuspid valves which formed two cusps, which I showed to the Society for the Study of Disease in Children.‡

The septum may be absent, as in the cor trilobuläre, and as in a case shown by Ernest Clarke,§ where the right auricle rested on the thickened wall of the single ventricle, and where the pulmonary artery and aorta were one tube and did not bifurcate until $\frac{1}{2}$ in. beyond their combined origin. Or the septum may be represented by a falciform ridge springing from the lower and anterior wall of the ventricle. Or irregular defects may arise in it.

* 'Path. Soc. Trans.,' 1885, vol. xxxvi, p. 175.

† *Ibid.*, 1879, vol. xxx, p. 266.

‡ 'Reports of the Society for the Study of Disease in Children,' vol. viii, p. 340, pl. ix.

§ 'Path. Soc. Trans.,' 1885, vol. xxxvi, p. 178.

Changes in the heart muscle do not necessarily follow septal defects. In some cases, however, hypertrophy and dilatation of both ventricles are seen, and this is especially likely to occur on the right side. In my child,* aged 2 years, the right ventricle was hypertrophied, the left normal. In the infant aged 3 months there was no hypertrophy, but in the infant aged 8 months there was hypertrophy and dilatation of the right ventricle.

In these three cases in regard to cyanosis, the child aged 2 years was born a "blue baby," but she was not blue; in the infant aged 3 months the lips were of good colour, the face pale; in the child aged 8 months the lips were rather lilac coloured when crying, but in this infant there was a patent ductus arteriosus also. According to Maude Abbott,† of 32 primary defects collected from the literature, one of which included a case of my own,‡ cyanosis was absent in 16, slight in 8, moderate in 3, and marked in 3. Cyanosis is usually marked where there is deviation of the aorta to the right. In primary septal defects the pulmonary artery is apt to be dilated, and this was noticed in two of my children.

Defects in the septum do not always produce murmurs, but when bruits are present the following characteristics have been noticed by me in the pure cases which have come under my observation. In the child, aged 2 years, there was a loud rough systolic bruit audible all over the chest, the point of maximum intensity being over the pulmonary area. It was not audible in the big vessels of the neck; the second pulmonary sound was accentuated. In the infant, aged 8 months, the systolic bruit was heard loudest at the junction of the xiphoid with the gladiolus on the left, and it was conducted a short way into the axilla. A faint systolic bruit was heard only occasionally at the left base. It was not transmitted to the neck-vessels. The second pulmonary sound was accentuated. In the infant, aged 3 months, there was a loud systolic murmur at the left base, which was traceable as far as the left mid-axillary line. The second sounds were normal, and in this infant the heart was not enlarged. In the oldest child there was a marked systolic thrill all over the cardiac area, and in the child aged 8 months a systolic thrill was occasionally felt at the left base, but this might have been due to the patent ductus arteriosus, and in the infant aged 3 months it was absent.

(To be continued.)

* 'Reports of the Society for the Study of Disease in Children,' vol. vi, p. 241.

† 'System of Medicine' (Osler and McCrae), vol. iv.

‡ 'Reports of the Society for the Study of Disease in Children,' vol. vi, p. 241.

TREATMENT OF ADVANCED CASES OF ACUTE
DIAPHYSITIS.

By H. M. W. GRAY, M.B., F.R.C.S.Ed.,

Surgeon and Lecturer on Clinical Surgery, Aberdeen Royal Infirmary.

G. M—, female, aged 11 years, was admitted to hospital on March the 31st, 1908, suffering from acute suppurative diaphysitis or osteomyelitis of the upper half of the shaft of the left tibia. The history and clinical symptoms were typical of such a case. The upper epiphysis and neighbouring knee-joint were, so far as could be ascertained, free from disease. The child was at once etherised and the limb thoroughly disinfected. An incision, slightly internal to the crest of the tibia, was made from the upper limit to $\frac{3}{4}$ in. below the lower limit of denudation of periosteum, down to bone in its whole extent. The subperiosteal cavity was washed out, first with peroxide of hydrogen (10 per cent.) and then with saline solution. The periosteum, corresponding in extent with the lower $\frac{3}{4}$ in. of the incision, which was adherent to bone and healthy, was elevated off the bone, which was then chiselled across at the lower end of the incision. The upper half of the tibia was then easily lifted out of its periosteal bed, as it was only very loosely adherent to the epiphysial cartilage. The cavity thus made was carefully washed out as before, dried, and packed lightly with sterile gauze wrung out of peroxide of hydrogen (3 per cent.). The opening of the medullary cavity of the remaining part of the tibia was previously smeared over with Beck's bismuth paste, so as to prevent any possibly infective material from penetrating to the healthy medullary cavity of the lower part of the bone (Fig. 1). The limb was then enveloped in sterile dressings and a moulded splint applied (made of plaster-of-Paris bandages soaked in biniodide of mercury [1 in 2000] solution).

The temperature fell to normal in a few hours. Three days later the child was anaesthetised again and the gauze was removed. The cavity was found to be clean and dry, free from pus. Thin catgut interrupted sutures were passed through the edges of the periosteum and left untied. Beck's bismuth paste was poured into the cavity, allowed to set partially, and then the catgut sutures were tied. Bismuth paste which oozed out between the stitches was lightly wiped away and the skin was carefully sutured with fine catgut. Dressings were applied and the limb placed again in the plaster splint.

The subsequent history is of most interest in connection with the

rapid and practically complete regeneration of bone which took place. This is seen by referring to Figs. 2, 3, and 4, which represent skiagrams taken respectively fifteen days, six weeks, and thirteen months after operation. Small sinuses formed, one at the upper and one at the lower end of the wound. Through the upper sinus the greater part of the bismuth paste was gradually pushed out—as the



FIG. 1.—Skiagram taken April the 1st, 1908. Absence of any shadow corresponding to the periosteal tube of the upper half of the diaphysis of the tibia. Shows the mass of bismuth paste closing the medullary cavity of the lower half.

periosteal cavity became filled with “granulation tissue” and bone. The discharge was a thin, honey-like fluid. At no time was there an ordinary purulent discharge. The lower sinus was found to be due to the presence of a thin flake of necrosed bone, which separated and was extruded during the sixth month. At the end of this month the patient was able to walk freely without pain. At the present time, thirteen months after operation, she has perfect use

of the limb, has no discomfort of any kind, and on examination, apart from the scar, one can make out scarcely any abnormality of the bone. On careful measurement from the upper margin of the internal tuberosity to the tip of the internal malleolus it is found that the affected tibia is $\frac{1}{4}$ in. shorter than the other. It was



FIG. 2.—Shows slight growth of periosteal bone. The dark shadow is a mass of bismuth paste. Skiagram taken April the 15th, 1908.

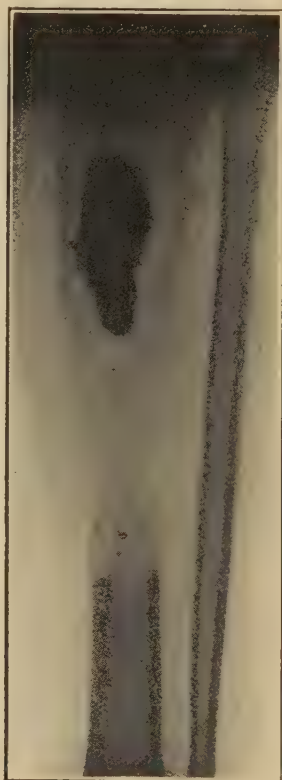


FIG. 3.—Shows greater growth of periosteal bone. Smaller amount of bismuth paste than in Fig. 2. Skiagram taken May the 14th, 1908.

thought that the girl had grown four or five inches during the past year. Unfortunately measurements were not taken when she was in hospital.

One may quote, as being representative of sound modern surgical procedure in such cases, what is said in Keen's 'Surgery,' vol. ii, pp. 37-40: "Treatment of the acute stage: In the acute stage there is suppuration of the marrow, more or less extended through-

out the shaft, with often a subperiosteal abscess and perhaps abscess of the soft parts. The indications are the same as in any other acute suppuration, *i. e.* the pus must be evacuated and the bone cavity must be drained. This demands not only an incision into the soft parts, but an opening into the shaft of the bone. For evacuation of the bone at first a good sized trephine button, $\frac{1}{2}$ in. in diameter,



FIG. 4.—Shows leg-bones of both sides. The skiagram on right shows practically complete regeneration of tibia—no medullary cavity apparent yet. Note epiphysial “line.” Taken May, 1909.

of cortical bone should be removed. If pus escapes from the marrow, this trephine hole should be enlarged by chisel and gouge, and should be extended along the shaft so long as definite pus escapes from the marrow. . . . Treatment of the subacute stage: After the acute process has subsided, either from spontaneous or artificial evacuation of the pus . . . the treatment . . . may be divided into three classes: (1) Removal of the

necrotic sequestrum before a definite involucrum has been formed, while the periosteum, although proliferating, is still plastic; (2) removal of the sequestrum just as soon as a sufficient amount of involucrum has been formed to carry on the function of the original shaft. In the early stages such a young involucrum has a limited power of central growth, and in favourable cases may obliterate the cavity left by the removal of the sequestrum. (3) Removal of the sequestrum after the involucrum has become dense bone, like ordinary cortical bone. . . . Removal of sequestrum while periosteum is plastic. . . . The time for this operation is when the periosteum has begun well-marked ossification in its deeper layers, and yet ossification is not so far advanced as to form a rigid periosteal shell. The time varies somewhat in different cases, but the average time is about the eighth week after the acute infection has been stopped by evacuation of the pus."

The treatment which I have detailed above in the description of an illustrative case is in advance of the treatment just quoted because (1) time, to the extent of at least two months, is saved to the patient; (2) the focus of infection, and, at the same time, a cause of continued irritation and suppuration is *completely* got rid of at the first operation (this may sometimes turn the scale against a fatal issue); (3) multiple operations are dispensed with. The manipulations carried out under anaesthesia when the original gauze plugging is removed do not involve any strain on the patient's powers of recovery.

What is exactly the best material with which to fill the periosteal cavity is not certain, nor is it, indeed, certain that such internal support for the periosteum is necessary at all. In the case of a thin bone like the fibula it certainly is not necessary, as one of my cases showed. In the skiagrams, Figs. 2 and 3, one can see that the periosteal tube is not supported by any internal mass of bismuth paste in the lower part, yet in Fig. 4 one sees that the bone has been reproduced almost as thoroughly in this part as in the part above where the internal support was present.

If such material *is* found to be necessary, I have the feeling that some plastic mass, such as Beck's bismuth paste, is preferable to a more solid, less extrusable substance as, for example, plaster-of-Paris. If infection of the periosteal cavity recurs, such a foreign material as gypsum acts in the same way as a "natural" sequestrum and has to be removed in the same way. The mildly antiseptic, semi-solid bismuth paste has the advantages of preventing infection, of being easily extruded by the growing tissues, and of being more absorbable.

This method of treatment does not interfere with the function of the epiphysial cartilage. An equally good result was obtained in another case where the whole shaft of the tibia was affected and was removed. Even although both epiphysial cartilages were exposed by this removal yet after a year there was no appreciable shortening of the limb.

In cases where there is only a single long bone in the part affected (femur, humerus) it is probably wiser to follow the lines laid down by Dr. Nichols in Keen's 'Surgery' (vol. ii, p. 41) and wait, before removing the shaft, until the periosteum is rigid: "The difficulty of judging when this time has arrived is considerable, and can best be judged by means of the X ray."

I have called such cases as these under discussion "advanced" cases of diaphysitis. The inflammation should be nipped in the bud at the stage which some would call "acute epiphysitis," obviously an erroneous term because the focus begins nearly always in the diaphysis. When the pathology and symptoms of the incipient stage of the usual suppurative "osteomyelitis" become more understood, and when there is greater appreciation of what the simple modern treatment of this stage entails in the saving of time, trouble and danger, so will such cases become uncommon. Timely use of drill, burr, fine chisel or gouge at the tender spot in the end of the diaphysis will sometimes save a limb or a life, and in any case will prevent a prolonged illness.

FOUR CASES OF ACUTE MENINGITIS IN CHILDREN.

By FREDERICK TRESILIAN, M.D., F.R.C.P.Ed.

THE four cases, of which I have given a description below, offer, by their comparison, an opportunity of studying the different types of one of the most interesting affections in childhood. As they occurred in private practice no opportunity of a post-mortem examination was possible in the one case that died; and no means was available of diagnosis by lumbar puncture. Though they had much in common the types were distinctly different, and the symptoms and signs were peculiar in each case.

The first was one of tubercular meningitis, which began in a hemiplegic form and which at first took me in, for reasons which will be given.

CASE 1.—A female child, aged 4 years, seen on September the 20th, 1904. She was quite well until September the 20th, when, on getting up from a chair, she fell, and her mother perceived that she could not stand on her left leg, which doubled up if she tried to put weight on it. During that morning, also, she was sick two or three times. She complained that she did not feel well, but of nothing else definite.

This September was warm weather. An interesting point was that her elder sister, when about two years old, had infantile paralysis about four years previously, the attack occurring in July, which had left paralysis with wasting and shortening in the left leg.

When I saw the second child in the afternoon of September the 20th she seemed quite well, except that she could not move the left leg, which was limp and flaccid, and there was no knee-jerk or plantar reflex. I thought that it was possibly another case of infantile paralysis, but could find no evidence of affection of any other limbs or muscles save in the left leg, which was somewhat unusual.

Next day, September the 21st, there was similar flaccid paralysis with loss of knee-jerk and plantar reflex, and she complained of pain in the leg and back; there had been no sickness. Temperature was normal; pulse quick but regular; respiration normal. No headache; no cerebral symptoms, save picking at her lips, which was suspicious.

The condition remained the same until September the 24th to 25th, when in the early morning she awoke delirious and much worse. I saw her early and found irregular breathing and some dyspnoea. Pupils were dilated; temperature normal; pulse quick and feeble. The condition of the leg was the same, with absent knee-jerk and plantar reflex, both of which were present on the right, but not so brisk as on the previous day. There was no Kernig and no head retraction; she had incontinence of urine; there was flaccid paralysis of left arm. The bowels were constipated; temperature 101° – 102° F.

On September the 27th, three days later, the condition of arm and leg were the same; the pupils were dilated and inactive to light. There was some slight stupor; incontinence of urine. She died that same day, being actually comatose only for a few hours before death. There were no convulsions all through.

This must have been a case of tubercular meningitis of an acute type. I could find no evidence of tubercles of choroid, though I looked each day for them, which would have been the one point necessary to make the diagnosis of the condition absolute. There was no cardiac lesion to cause embolism, no rigors, and even no

pyrexia until the end, which is very unusual in any form of meningitis. I could not obtain a necropsy.

I have seen tubercular meningitis occur before in a somewhat hemiplegic form, but there have usually been convulsions somewhat of the Jacksonian type leaving a paresis after them.

CASE 2.—This was an ordinary case of posterior basic meningitis in an infant, which left behind it the very unusual sequela of complete deafness only.

A female baby, aged 8 months. Illness began on September the 11th, 1905. She was restless and would not sleep, and she had a convulsion that day. She was teething and had cut her teeth irregularly, the upper lateral incisors coming through first and then the upper right central incisor. The left upper central incisor was close to eruption and the gum was tense, tender and swollen. She had no more convulsions, but the next day she was very restless and strange and twitched a great deal. She was sick also on the first day. The child was well nourished and healthy, with no appearance of rickets. She had been fed on the breast. Action of bowels was regular.

On September the 16th she got retraction of the head markedly and she resented any effort to put it forward. Eyes were opened and staring, no photophobia, and she had convulsive tonic spasm of the hands. She was very restless and slept badly.

September the 17th: Head much retracted, cough and dyspnoea. Bronchitis at backs of lungs; no consolidation or evidence of pneumonia. Temperature 102° F. Knee-jerks normal, no Kernig. I lanced the gum over the left lateral incisor and gave liq. hydrarg. perchlor. five minims every three hours with inunction of mercury ointment into neck and occiput.

They had lost a child, aged 5 years, from tubercular meningitis four and three quarter years before. I attended her. This child had apparently had basic meningitis when three and a half years old, and was absolutely deaf after it. She had just learned to talk, but after this illness her talking gradually went, and though she never became quite a deaf-mute, she gradually lost what little speech she had acquired.

September the 22nd (11th day): Child is much better. Head retraction has almost gone; she looks about her much more and sleeps well. Upper left central incisor is well through and the left lower central incisor has also erupted.

The next note is January the 31st, 1906: The mother brought the baby, having discovered that she was deaf. I thought, however,

that the child could hear a Galton's whistle and tuning-fork in the air when close to the ears. The eyes looked normal.

February the 17th, 1906: The child looks bright and well. I could not be at all sure that she heard a Galton's whistle or tuning-fork by air-conduction, and she did not pay much attention to a tuning-fork held on the skull. I have seen the child occasionally since, and there is no doubt that she is absolutely deaf. An interesting point is as to whether the deafness is to be attributed to the effects of the meningitis or to destructive effects of the initial convulsion. If due to the meningitis, it would be the effect of lesions of the trunk of the nerve above the internal auditory meatus. If due to the convulsion it would be central, meaning a destructive lesion of the nerve-cells in the occipital lobes and angular gyrus. The symptoms in either condition would be identical, and the same as those of an acute destructive lesion of the labyrinth.

The next case was a typical and severe one of acute cerebro-spinal meningitis.

CASE 3.—A. H—, male, aged 11 years. Illness began suddenly on March the 8th, 1903, with repeated vomiting, stomach pain and rigors, followed by intense headache and backache. There was slight diarrhoea at the onset, which soon ceased, and constipation ensued. The sickness continued for two days.

The boy complained of intense vertical and frontal headache, and of severe pain in the back when he moved, all up the spine. Last night he got out of bed and came into his mother's room complaining of these pains. Pulse was 120; temperature 102° F. Tongue furred; skin hot and dry. Occasional internal strabismus.

March the 15th: Symptoms about the same; headache, chiefly frontal, severe, and paroxysmal. Temperature lower; double optic neuritis.

March the 18th: Temperature normal. Optic neuritis less severe. Kernig's sign present on both sides. Mental condition clearer. Knee-jerks feeble but present.

March the 21st: Admitted to Hospital. Condition the same. Phenacetin relieves his head. Gave hyd. $\bar{c}$ creta.

March the 27th: Severe paroxysms of headache still occur, chiefly frontal. Sleeps occasionally. Takes food well. Superficial reflexes very exaggerated with hyperæsthetic condition of the skin. Kernig positive on both sides. Rigidity of legs, especially right one. Knee-jerks absent. Flexor response on both sides.

April the 3rd: Headache intense and more constant. Optic neuritis still marked. I gave liq. hydrarg. perchlor. 3ss *t.d.s.* and ice-cap to head.

April the 10th: Still intense headache. Kernig and optic neuritis still present. Tongue cleaner. Rigidity of legs. Weak action of left external rectus.

April the 16th: Temperature fell to normal and remained so. Marked improvement began and continued.

May the 13th: Knee-jerk present on the left side and a remnant of a Kernig on same side. No knee-jerk on right side. Rigidity much less. General condition good. From this time on he convalesced and did well.

His optic discs recovered perfectly with no subsequent atrophy, although the severity of the neuritis closely approached that of a "choked disc."

I feel quite confident of the therapeutic effects of the mercury given on April the 3rd; improvement set in so soon afterwards, with decline of temperature and gradual disappearance of the characteristic signs of the disease.

CASE 4: *Posterior basic meningitis*.—No exact dates taken. Male child, aged 3 years. Illness came on with headache, sickness and fever, crying out, and finally convulsions, followed by stupor and coma and marked head retraction. The screaming was incessant and somewhat like that of tubercular meningitis. There had been no causal head injury and there was no ear disease. The eyes were open and staring with dilated pupils inactive to light. No photophobia. There was a spastic paralysis of the right arm, which was rigid with the fingers tightly clenched. A bed-sore formed on the sacrum; there was constipation, and urine was passed involuntarily. Corneal reflex was absent. The knee-jerk was absent on the right side. The eyes were frequently but not constantly deviated to the right side. This comatose condition continued with partial remissions of a few hours for fourteen days, during which time there were frequent attacks of convulsions, which were general and bilateral, and head retraction was marked during the same period. There was no history of tubercle in the family. The treatment consisted in shaving the head and constant application of ice. Blisters on mastoids. Inunction daily of ung. hydrarg. with equal parts of vaseline, and liq. hyd. perchl. mv every four hours. The gums became white and spongy and salivation occurred. The child improved and ultimately got well in about a month. Coma disappeared; pupil reflex returned. Spasm of arm disappeared, and convulsions decreased in frequency and severity. During convalescence his temperature ran up suddenly to 103° F., but a few doses of calomel set it right. There was no relapse of original symptoms and recovery was complete.

These four cases offer good illustrations of the forms of meningitis occurring in infancy otherwise than ordinary tubercular. They exhibit altogether all the signs and symptoms met with in non-tubercular meningitis, and even though they have many points in common there are distinctions peculiar to each case. I cannot help thinking that they show in a marked degree the usefulness of mercury in the treatment of meningitis. I believe in it firmly, and in the last case I stuck to it right through without change or cessation in what seemed to be a hopeless case. This was one of the few cases I have ever given mercury to produce salivation. Its absorbing powers are marked, and are frequently demonstrated in its application to tubercular peritonitis and enteritis and in ocular diseases. I recently saw a girl who looked as if she was moribund and hopeless from tubercular enteritis, and who got quite well under daily applications of unguent. hydrargyri. It might be used with advantage as an external application in cases of serous effusion in pleurisy, tubercular or otherwise.

I trust these short notes will be evidence not only of the great clinical interest to be found in meningitis, but also will impress the fact that, like pneumonia, meningitis is a disease worthy of pegging at vigorously, and that in mercury one has a drug of a quite decided value in the treatment of the disease.

The Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Meeting held in the Scotch Capital, Dr. George Carpenter, Vice-President of the Society and Chairman of Council of the Section, presiding.

THE Annual Meeting in Great Britain of the Children's Section of this Society was held at Edinburgh on June the 18th. The members of the Section were received with cordial hospitality by the medical profession in Edinburgh, and were entertained to lunch before the meetings.

At the meeting in the afternoon there was first a discussion of cases. Tea was then served, after which papers were read. On the following day a number of extremely interesting cases were shown at the Edinburgh Royal Hospital for Sick Children to those members who remained in Edinburgh.

CASES.

A Case of Patent Ductus Arteriosus in a Young Woman was shown by Dr. G. A. GIBSON.

The CHAIRMAN (Dr. GEORGE CARPENTER) said that it was seldom such cases were seen clinically. In this condition there might be a purely systolic

murmur and not a double murmur. He suggested that an X-ray photograph should be taken of the case, as it had been shown that patent ductus arteriosus could be recognised by this means. The malady was not infrequently associated with other congenital somatic malformations.

A Case of Delayed Rickets was shown by Dr. BURNET.

Dr. G. A. SUTHERLAND asked if the rickets had developed recently, or if they had persisted from infancy.

Mr. A. H. TUBBY thought that rickets was a disorder beginning in childhood, and continuing with outbreaks at intervals afterwards, the causes of which are unknown.

Dr. SPRIGGS referred to recent work on exercise as a cause of rickets.

Dr. Macalister, Mr. Harold Stiles, and the Chairman (Dr. George Carpenter) also discussed the case.

A Case of Deformity of the Cervical Spine and the Right Scapula, in which there was a fold of skin at the side of the neck reaching from the head to the shoulder, was shown by Mr. SCOTT CARMICHAEL.

Mr. TUBBY said it was one of those curious cases known as Sprengel's shoulder. The right scapula differed in shape from the left. The movements of the shoulder were limited. He also suspected the presence of supernumerary cervical ribs. The skin folds, which he had not seen before, showed, he thought, the traction of the skin in the attempted descent of the scapula.

The CHAIRMAN (Dr. GEORGE CARPENTER) asked if there were any other deformities, and—

Mr. TUBBY said that in about one third of the cases congenital heart disease was associated.

A Case after Cholecystenterostomy for Traumatic Obstruction of the Common Bile-Duct was shown by Mr. HAROLD STILES.

The CHAIRMAN (Dr. GEORGE CARPENTER) suggested that, as this case had been so successful, congenital malformations of the bile-duct could probably be treated surgically.

Dr. JOHN THOMSON said that cases of congenital malformation of the bile-duct had been operated on, but the result had always proved that the operation could not possibly have been expected to do good, as it is not a local condition but a general disease.

Dr. SPRIGGS suggested that metabolic observations should be carried out in this case, as the bile ran straight into the large intestine, and it would be interesting to see how the digestion of fats was affected by the absence of bile from the small intestine in man.

Mr. HUGH LETT referred to a case of traumatic obstruction of the bile-duct which recovered.

The CHAIRMAN (Dr. GEORGE CARPENTER) said that operation might be successful in congenital cases in which the stenosis was merely related to the common duct.

Two Girls in whom the Ureters were Transplanted into the Pelvic Colon for Incontinence due to Epispadias without Extroversion of the Bladder were shown by Mr. HAROLD STILES. The operation had relieved the incontinence, and the kidneys had not been affected by the transplanting of the ureters into the colon.

Two Cases of Hydrocephalus were shown by Mr. HAROLD STILES, and

in each of them both common carotids had been ligatured with the object of lessening the secretion of cerebro-spinal fluid, with favourable results.

Dr. G. A. SUTHERLAND said that they all appreciated any new method of treatment of this disease. In the congenital cases there was usually a continuous flow of cerebro-spinal fluid and no blocking. It was possible to decide whether blocking of the fluid was present by means of lumbar puncture. Certain cases of syphilitic origin recovered under mercurial treatment. The special type where this operation might do good would be where there was a slight increase of secretion over absorption. Mr. Stiles had spoken very guardedly of results, but with these successes he might certainly be encouraged to continue. Dr. Sutherland referred to his former work on subdural drainage, and said he still believed in it, and some patients had certainly been cured by it.

Mr. TUBBY said that these cases were the most successful he had seen.

Dr. SPRIGGS said that Mr. Pendlebury had suggested draining the ventricles direct into the subcutaneous tissues by carrying silk directly from the ventricles, after the manner of Handley's operation for œdema of the extremities.

Dr. SUTHERLAND said that in drainage methods the great difficulty was in keeping open the passages through the cerebral tissues.

Dr. JOHN THOMSON said that in several cases he had seen a very rapid cessation of increase of the size of the head after this operation. In some cases it had done no good. The head sometimes ceased to increase without operation. He thought the operation a justifiable one.

Mr. R. H. PARRY asked if it had been performed when there was spina bifida present also.

Mr. STILES, in reply, said that his first two cases had been most successful. It was difficult to get suitable cases, and the children were often very frail and delicate. He had never seen any benefit when the cases were complicated with spina bifida. Hydrocephalus was more liable to occur after the spina bifida was cured, when the spina bifida had been excoriated or ulcerated. Congestion may be set up from the region of the spina bifida, which passes to the fourth ventricle and sets up a true secondary hydrocephalus.

Cases illustrating Resection of the Diaphysis of the Long Bones for Diffuse Tuberculous Osteomyelitis was shown by Mr. HAROLD STILES. The long bones, for instance the tibia, which had been excised sub-periosteally, were exhibited, whilst on the couch lay the patient, in whom a new tibia had grown.

Mr. TUBBY congratulated Mr. Stiles on his excellent results in these cases, which he regarded as a most important piece of work.

The CHAIRMAN (Dr. GEORGE CARPENTER) expressed a vote of thanks to the Edinburgh gentlemen who had shown cases and for giving them a most instructive afternoon. Cases had been shown which it would have been worth while coming three times the distance to see. He hoped that it might lead to further co-ordination between Edinburgh and London in the study of disease in children.

Mr. HUGH LETT, in seconding the vote of thanks, heartily endorsed the Chairman's remarks.

PAPERS.

On Symmetry and Asymmetry and their Effect in the Production of Lateral Curvature of the Spine, by Mr. A. TUBBY, London.

Mr. PARRY asked if it was Mr. Tubby's experience that the left limb was more constantly longer than the right.

Mr. TUBBY thought not.

Observations concerning the Blood in Chorea and Rheumatism, by Dr. C. J. MACALISTER, Liverpool. In experiments made upon the length of life of leucocytes in the blood-plasma of patients suffering from chorea and rheumatism, he found that when the corpuscles of a chorea patient were placed in the plasma of another chorea patient they will live nearly as long as the corpuscles of a healthy person will live in another healthy person's plasma, showing that to some extent the blood-cells have become immune to the toxin. This being so, if the toxin in acute rheumatism is similar to that in chorea, the same evidence of immunity should exist when the corpuscles of the rheumatic patient are placed in the plasma of a patient suffering from chorea. When this experiment was carried out, however, it was found that the lives of the cells were invariably shortened, and one may infer from this that at all events some difference exists between the poisons in the two conditions. Dr. Macalister also supported the observation, previously reported by Cabot, that a marked eosinophilia is present in the subjects of chorea, whereas in acute rheumatism he did not find any increase of eosinophile leucocytes. His observations confirmed the suspicion that there is less real association between chorea and rheumatism than is generally thought to be the case, and therefore that chorea may be due to infective toxæmia of a distinctive character.

On a Case of Multiple Abscesses of the Liver in a Child, secondary to Appendicitis, by Dr. R. G. McKERRON, Aberdeen.

Mr. LETT referred to the difficulty of diagnosing between subphrenic abscess and appendicitis where there was jaundice, a little swelling of the liver and gall-bladder, and a history of prolonged pain and swelling in the epigastrium. If diarrhœa, vomiting, and distension of the abdomen were present, appendicitis was the most probable cause of it.

On the Ætiology and Treatment of Hyperpyrexia in Children, by Dr. JAMES BURNET, Edinburgh. Two cases were described. The first was in a child aged 7 years, with a tuberculous family history. The temperature reached 107° F., and was effectively treated by pouring cold water over the head and neck from a jug. The child was well next day, and no cause was discovered. The second was a boy, aged 8½ years, suffering from acute rheumatism, who was being treated with ten-grain doses of sodium salicylate. The temperature rose to 107.2° F. The same treatment was adopted, followed by the application to the head of sponges wrung out of iced water. The patient made an excellent recovery. The hyperpyrexia in this case was ascribed to the development of pericarditis, though Dr. Burnet thought that the salicylates might have something to do with it, especially as the high temperature was accompanied by a maniacal state. He was doubtful as to the safety of applying an ice-bag to the chest in such cases, for this might produce a great deal of shock and perhaps collapse. He recommended tepid sponging in the first instance, followed by the application of cold to the head by sponging or ice-bag. Should these measures fail the cold douche over the head and neck should be used, the head being held over a basin or bath.

The CHAIRMAN (Dr. GEORGE CARPENTER) said he had used ice-bags in

pneumonia and found that they were borne well, except when placed over the heart, when the children collapsed at once.

Dr. SPRIGGS said he used ice poultices. They should be made quite thin with gutta-percha tissue because of the pressure. He thought that the harm done by an ice-bag over the heart in children was due to the weight. No weight of any kind should be placed on the front of the chest in a child when it was ill, owing to the impairment of respiration which was brought about mechanically.

Dr. McKERRON had not seen collapse produced by treating pericardial affections with ice.

Dr. SIMPSON said that children who liked it could keep it on very well, but in other cases it caused great discomfort. He emphasised the necessity of keeping the child packed in hot bottles while putting on the ice-bag.

On a Case of Acute Leucocythæmia in a Child aged 8 years, by Dr. C. P. LAPAGE, Manchester. The first symptoms of the illness were headaches, some wasting, and a slight discharge from the ear, with drowsiness and irritability if roused. After these symptoms had lasted for seven to eighteen days the child began to vomit and to complain of tingling and numbness of the fingers of both hands. There was no history of hæmorrhage, tonsillitis, diarrhœa, dropsy, fainting, or impairment of vision. On admission three weeks after the beginning of the illness the temperature was 100° and there were chains of enlarged glands in the axillæ, the groins, the anterior triangles of the neck, the glands being discrete and none of them larger than a pea. There was no œdema, otorrhœa, or exophthalmos. A slight degree of vomiting was present. The stools were loose, but showed no other abnormality. The spleen reached down to below the brim of the pelvis, and the liver reached to one inch below the costal margin. There were signs of consolidation at the base of the right lung. The reflexes were increased. Kernig's sign was present but not well marked. Babinski's sign was present on the right side. The blood-count gave reds 1,600,000, whites 432,000, Hb. 35 per cent., index 1.1. There was an enormous number of large lymphocytes. The blood was whitish in appearance, resembling blood mixed with pus. It was less coagulable than normal. Death occurred one week after the first symptoms. The spleen diminished in size during the last days of life. Post-mortem, that organ showed early fibrosis. The bone-marrow of the femur and sternum was red in colour, and that of the ribs apparently normal. Pneumonia was present in the lungs, and there were ecchymoses on the visceral surface of the pericardium, sections of which showed infiltration of lymphocytes. A terminal infection with streptococci and staphylococci was found.

On the Treatment of Irreducible Intussusception by Lateral Anastomosis, by Mr. R. H. PARRY, Glasgow. This method of treatment was said in the text-books to be only applicable to chronic cases. Two acute cases were referred to, one of Mr. Parry's and one of Dr. Rutherford's. On the tenth day, in Mr. Parry's case, no trace of a tumour could be felt, and this was verified by examination. Mr. Parry put forward, firstly, the simplicity and safety of the method as compared with excision; secondly, that an alternative line of treatment was afforded to that of severe manipulation in irreducible intussusception; and thirdly, the disappearance of the tumour.

Mr. HAROLD STILES said it was a most instructive paper, and a landmark in the treatment of irreducible intussusception. He had thought a good

deal about the subject and had become almost pessimistic. Patients had died after resection where he thought recovery would take place. He had been agreeably surprised that a good many cases had recovered where there had been extreme difficulty in reducing the intussusception and even after splitting the peritoneal coat. The gangrenous cases were very difficult, and his only criticism of the paper was that the treatment was hardly applicable to those. He said he had had better results when he operated as quickly as possible, and though the children often looked moribund it was wonderful, if the treatment was properly carried out—no drugs and plenty of saline by the bowel—how they recovered. Once they had got over the shock he was no longer anxious about them. He thought this method of Dr. Parry's was very sound, and he would certainly try it in cases where there was a good deal of difficulty in the attempt to reduce the intussusception.

Mr. GEORGE CHIENE said if the intussusception could not be completely reduced owing to the œdema, a way of getting over the difficulty was by wrapping the portion in three or four large pieces of gauze and compressing it for three or four minutes. It was perfectly easy to do, and the child made a good recovery.

Dr. PARRY, in reply, said that with wider knowledge and means of early diagnosis these difficult cases would not be so often met with, and the treatment would resolve itself into reduction in the simple way.

On the Primary Source of Infection of Glands in the Neck, by Mr. E. SCOTT CARMICHAEL. By cutting sections of removed tonsils he had found tuberculous foci in a large proportion of cases.

Mr. TUBBY had not discovered tuberculosis in the tonsils so often. It would be interesting to know how often the tubercle bacilli were found in adenoids, and in the different tissues of the mouth,

Mr. PARRY referred to work on the matter by Dr. Nicholl, of Glasgow.

Mr. LETT felt convinced the source of infection was often found in the tonsils, and the operation did not seem complete if the focus was left behind.

Mr. STILES said that in those rapid forms of tuberculosis of the glands where the whole set of glands was involved in a few weeks one often found the tonsils were infected. Even if three quarters of the tonsil were removed the healing process destroyed the rest of the disease.

The CHAIRMAN (Dr. GEORGE CARPENTER) moved a vote of thanks to the management of the Royal Edinburgh Hospital for Sick Children for the use of the hospital, and he asked Mr. Stiles to convey their appreciation of the privilege.

Société de Pédiatrie, Paris.

May the 18th, 1909. Bulletin No. 5.

Dermoid Cyst of the Broad Ligament.—M. VILLEMEN showed this cyst, which had a twisted pedicle, removed from a girl, aged 5 years and 2 months, who had suffered for four months from pain, vomiting, and rise of temperature, followed by diminution of the swelling and pains. There was a tumour in the right iliac fossa suggesting appendicitis or ileo-cæcal tuber-

culosis. The tumour was adherent to the cæcum, mesentery, and uterus. The ovary was free and healthy.

Treatment of Whooping-cough by Injections of Morphine.—M. COMBY gave full details of six cases; one did not improve, one improved, and four were uninfluenced by the treatment. He was of opinion that, generally speaking, these injections diminished neither the intensity nor frequency of the attacks, nor did they check vomiting nor shorten the duration of the complaint nor prevent complications. One of the cases died of broncho-pneumonia.

M. MARFAN had treated in this way eighteen cases. In four the records were unserviceable as the children had for several reasons not been sufficiently treated. Observations on the remaining cases led to the conclusion: (1) That the tolerance displayed was extraordinary, doses from $\frac{1}{3}$ to $\frac{3}{4}$ cgrm. being given; only one child was drowsy, but his urine contained 1 grm. of albumin and he died of broncho-pneumonia ten days after the second injection. (2) In ten cases the attacks were notably diminished, in three cases the result seemed doubtful, and in one case no effect was produced. (3) The effect on the general condition was good, the children being bright and had good appetites. (4) The effect on vomiting was good in spite of the ordinary emetic action of morphine. (5) The effect on tachypnoea and tachycardia was equally beneficial. (6) The duration of the disease seemed lessened, although it was difficult to be sure on this point. He was of opinion that it should not be used as a routine treatment, but in a case where the attacks were violent, prolonged, frequent, followed by vomiting and causing exhaustion he would not hesitate to have recourse to injections of morphine.

M. TRIBOULET insisted on the advantages of this treatment in severe forms of the disease; it caused no gastric disturbance as did belladonna, bromoform, etc. He was pleased to find his own experiences corroborated by those of M. Marfan, but more observations were necessary.

M. BARBIER said it was difficult to come to any definite conclusion amid the conflicting statements of MM. Comby and Marfan. He did not accept in principle the treatment of whooping-cough by morphine, but preferred expectorants. He feared that the action of morphine, like the majority of really active substances, only acted unfavourably in this disease.

M. VARIOT said that clinical facts were of more value than *à priori* arguments in estimating the value of a new treatment, and those of M. Marfan had given interesting results, which confirmed the first communication of M. Triboulet on this subject.

Note on the Leucocyte Formula in Chickenpox.—MM. WEILL and ROULIER gave the result of their observations on twenty cases, and conclude that chickenpox is characterised by a normal leucocyte formula and by the absence of abnormal forms of white corpuscles, and that the hæmatologic characters of this disease present a striking difference to those of smallpox.

Hereditary Syphilis of the Lung; Death from a Gangrenous Focus in the Opposite Lung.—M. APERT showed post-mortem specimens which presented marked dilatation of the bronchial tubes, which were surrounded by bands of fibrous tissue. The nature of these lesions offered an explanation as to the uselessness of specific treatment, and in another case of the same nature, which the author showed before the Society two years previously, considerable improvement had followed subcutaneous

injections of gomenol oil, in series of a dozen, at intervals varying from a fortnight to two months.

Painful Hyperæsthesia in Typhoid Fever in a Child, aged 10 years.—MM. PASSEAU and TIXIER read notes of this case. Simple touching evoked painful sensations in the lower limbs and lower part of the trunk, while pressure and palpation, on the contrary, called forth no complaint. The condition lasted eight days.

Tuberculous Cavities in the Liver and Hydronephrosis.—The same authors also showed specimens from a girl, aged 5 years, who died after a few weeks with symptoms of general tuberculosis. The lesions found at the autopsy were of old standing—large vomicae throughout the whole of the parenchyma of the liver, and a large hydronephrosis of the right kidney, which was reduced to a mere shell. Lesions of such a marked character were not exceptional, but it was rare to find them associated in the same subject.

Case of Tubercle of the Thymus in an Infant.—M. TISSIER and Mlle. FELDZER read notes of a case in M. Hutinel's clinique of a child, aged 1 year, suffering from convulsions and wasting. There was contraction of the right arm, marked rigidity, together with Kernig and Brudensky's signs. Tuberculous meningitis was confirmed by lumbar puncture. Three quarters of the thymus were invaded by the tuberculous process in different stages of development, caseous granulations and cavities. Only the lower half of the right lobe had a normal aspect. The absence of general symptoms must be attributed to functional hyperactivity of this portion. In spite of the thickness of the gland (2 cm.) there were no signs of compression. Tubercle of this organ is so rare in children that its occurrence has been denied by several authors.

VINCENT DICKINSON.

Abstracts from Current Literature.

Medicine.

The epidemiology of acute poliomyelitis. A study of thirty-five epidemics (*Amer. Journ. of Med. Sci.*, 1908, p. 647).—L. Emmett Holt and Frederic H. Bartlett have collected reports of 35 epidemics of poliomyelitis prior to the year 1907. In 23 of the epidemics the number of reported cases is less than 20; in 7 there were from 20 to 50 cases; in only 4 were there over 100. These 4 were: Australia in 1904, 108 cases; Vermont in 1894, 132 cases; in all Norway in 1905, 719 cases, and in 1906, 334 cases. The seasonal occurrence is strikingly uniform. In 33 epidemics they occurred in the hot season only, the most frequent months being July, August and September. In almost every instance the epidemic terminated with the month of October. In one Australian epidemic the months of occurrence were March and April, which correspond to October and November in our climate. Location and surroundings seem to have little influence upon the occurrence of epidemics, which have been about equally divided between country and city. The mortality was difficult to obtain in sporadic cases, but in a study of the epidemics there were found to be 201

deaths in 1659 cases, a mortality of 12.1 per cent. The great majority of the persons attacked in epidemics were children under four years of age. Their conclusions as to the communicability of acute poliomyelitis were as follows: "The occurrence of epidemics and the relation of certain groups of cases to one another in these epidemics place beyond question the statement that acute poliomyelitis is an infectious disease. Whether we can go farther and state that the disease is communicable is an open question. After carefully considering all the evidence brought together in this paper, we cannot resist the conclusion that the disease is communicable, although only to a very slight degree, one of the most striking facts being the development of the second cases within ten days after possible exposure. Positive statements, however, must be deferred until the discovery of the infectious agent."

JAMES E. H. SAWYER (Birmingham).

Acute lymphatic leukæmia (*'St. Bartholomew's Hosp. Rep.,'* vol. XLIII, edited by H. Morley Fletcher and W. McAdam Eccles).—A boy, aged 3 years, was admitted to the hospital with petechial cutaneous eruption, hæmaturia and pyrexia. In the mouth was an ulcer involving the tongue, gum and cheek, with a greyish-black foul slough, which also filled the sockets of the teeth. These were extracted; the ulcer was curetted and swabbed with pure carbolic acid. The child's condition then improved. A month later the cervical glands began to enlarge rapidly, and other glands all over the body became enlarged and pyrexia returned. A week later the spleen was found to be palpable, the gums became spongy and bled freely. The blood-count showed red corpuscles 2,570,000; white cells 482,000; mononuclear cells 97 per cent. (large form 92 per cent., small form 5 per cent.); polymorphonuclear 2 per cent.; eosinophile cells, including myelocytes, 1 per cent. The child died a week later.

JAMES E. H. SAWYER (Birmingham).

Sarcoma of the palate (*'St. Bartholomew's Hosp. Rep.,'* vol. XLIII, edited by H. Morley Fletcher and W. McAdam Eccles).—A boy, aged 5 years, was admitted to the hospital with the history of a quinsy, which had been opened three or four months previously. A swelling was present over the hard palate and anterior pillars of the fauces measuring 1 inch by $\frac{1}{2}$ inch and containing a slough in a cavity $\frac{3}{4}$ inch deep. There was profuse bleeding. A portion of the swelling when examined histologically was shown to be a mixed-celled sarcoma. The glands in the neck became affected, and the child left the hospital without operation.

JAMES E. H. SAWYER (Birmingham).

Acute bulbar paralysis (*'Med. Press,'* February 3, 1909).—At the Gesellschaft of Vienna Popper showed a boy, aged 6 years, with acute bulbar paralysis, which had for its origin poliomyelitis. At one time the paralysis seemed to recede, and the cerebral ataxia as well as the nystagmus improved. Up to that time the unsteady gait, weakness of the facialis and paralysis of the ocular muscles had been absent as well as paralysis of the extremities.

T. R. WHIPHAM.

Concerning the presence of pepsin in the stomach of the nursling (*'Jahrb. f. Kinderheilk.,'* August, 1908).—Reeve-Ramsey, after a study of the gastric juice of healthy and abnormal infants, finds that pepsin is always present in the stomach of normal breast-fed children. In acute gastro-intestinal disturbances in nurslings it is also usually to be found. In

infants suffering from pyloro-spasm pepsin and hydrochloric acid may be present in greater amounts than in normal children of the same age. The stomach of children suffering from chronic atrophy frequently contains no pepsin, though if these atrophic children begin to improve and gain in weight the presence of pepsin can again be demonstrated. The gastric juice of normal healthy children is able to transform proteid into peptone, and this is possible without the addition of any acids other than those invariably found in the stomach. The pepsin in the gastric juice is able to do active digestion if lactic acid is present without hydrochloric acid. In some cases hydrochloric and lactic acids are found even though no pepsin be present, and *per contra* pepsin can be present in the gastric juice without the presence of either hydrochloric or lactic acid.

T. R. WHIPHAM.

Causes of death in whooping-cough (*Thèses de Lyon*, 1907-1908, No. 41).—**A. Sarda**.—Among 169 cases of whooping-cough admitted to Weill's service at the Hospice de la Charité, Lyons, between 1894 and 1907, twenty-one died—a mortality of 12·4 per cent. This figure is higher than that of cases treated at home, but compares favourably with the results obtained in other hospitals (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1907, p. 454). Fourteen deaths were due to broncho-pneumonia, which usually occurs at the height of the disease in the second or third week. Three deaths were due to tuberculosis, one to measles, and one to convulsions. The younger the child the worse the prognosis. Seventeen of the twenty-one fatal cases were less than three years old.

J. D. ROLLESTON.

Lumbar puncture (*Arch. of Pediat.*, 1908, p. 738).—**E. Burvill-Holmes** describes the technique of the operation. He does not think it necessary to count the vertebrae, but feels with the thumb-nail for the space between the fourth and fifth lumbar vertebrae, which is on the level with the iliac crests, and thrusts the trocar through the space below (Chippault's sacro-lumbar foramen). A "dry tap" does not necessarily mean that one has failed to reach the canal. The subarachnoid space may be obliterated by inflammatory products, thus shutting off the ventricles, or the fluid may be too thick from admixture with leucocytes to flow through the needle.

J. D. ROLLESTON.

Aberrant vaccinia (*Deutsch. med. Wochens.*, 1908, p. 1856).—**L. Leven**.—An unvaccinated woman was sent to Leven by a gynaecologist with the diagnosis of syphilis. Two large ulcers on the labia closely resembled hard chancres. The history, however, was not that of syphilis. The appearance of the lesions had been sudden and attended with shivering. The patient stated that three weeks previously she had wiped her vulva with a rag which had served as a dressing for her recently vaccinated child. Eight days later the lesions had appeared. Treatment consisted in the employment of lysol sitz baths, and the ulcers rapidly healed (*cf.* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1909, p. 36).

J. D. ROLLESTON.

Digestive disorders in rickets (*La Presse Médicale*, November 18, 1908, No. 93, p. 745).—**Prof. A. B. Marfan** says that two kinds of disorders of digestion are found in rickets: first, those which are prodromal or accompany the onset of the complaint, consisting generally of relapsing gastro-intestinal catarrh, more rarely spasmodic dyspepsia with repeated vomiting; and secondly, those which represent the dyspepsia of confirmed

rickets, and consist of a special form of atonic dyspepsia with distended abdomen. These two kinds do not, however, represent two consecutive phases of the same process, and the author asks whether this form of dyspepsia with large flaccid abdomen is not a concomitant of rickets, constituting an integral part of this disease. He thinks it does, since it is admitted that rickets can show itself merely by slight osseous deformities which are only recognised by those who look for them systematically. As to the digestive troubles which may precede the appearance of osseous deformities and accompany the first phase of the disease, they consist most often in relapsing gastro-enteric catarrh due to defective feeding and principally to badly managed bottle feeding. This defective alimentation, *i. e.* deprivation of mother's milk, artificial feeding, premature weaning, causes first of all digestive troubles and later on the osseous deformities of rickets. Prof. Marfan is of opinion that artificial feeding and heredity, acting singly, or more frequently associated, effect a predisposition which favours to a marked degree the action of the chronic toxi-infection. He insists that the form of dyspepsia with flaccid large abdomen is rarely absent in cases of rickets whatever be its origin. His explanation is that the relaxation of the intestinal and abdominal muscular tissue which results in dilatation of the intestine and large flaccid abdomen is only a part of the general muscular atony of rickets described by Hagenbach-Burckhardt and Bing; that the slight swelling of Peyer's patches and follicles is only a part of the tendency to hyperplasia which in rickety subjects is found in the hæmato-poietic and lympho-poietic tissues.

VINCENT DICKINSON.

The causes and symptoms of simple chronic ulcer of the stomach in children (*La Clin. Infant.*, January 15, 1909, No. 2, p. 48).—**Parmentier** and **Lasnier** made this important communication to the Soc. des Hôpit., December 18, 1908. Chronic simple ulcer of the stomach in a child is so rare that such authors as Wiederhofer and Kundrat doubt its existence, while Rokitansky declared he had never met with it under the age of 14 years. Brinton only found it twice in 226 autopsies below the age of 10 years. Only about twenty cases have been recorded. The seat of the lesion does not differ markedly from that which holds good in adults. In nine autopsies the ulcer was six times in the pyloric region, three in the lesser curvature, four on the posterior surface, either cardiac or pyloric, once on the anterior surface near the cardia and once on the greater curvature. More than one ulcer was present in some cases. In sixteen cases, seven were boys and nine girls, aged from $2\frac{1}{2}$ to 14 years. As to probable causes, bad feeding and acute gastritis take the first place. In the authors' case the girl had measles and chickenpox between the age of 3 and 4 years, and gastro-enteritis in her first year. She was badly nourished, took only five feeds daily, and at the age of 9 months was put on cow's milk, which soon caused a diarrhœa which lasted several years. At two years broth was given in addition and a few weeks later she took what the others ate, *café au lait*, vegetable, stewed and boiled meat. Ulcerations due to swallowing caustics are not rare in children, and a foreign body, such as a coin, may produce the same result. Such ulcers may pass into a chronic form. The symptomatology is relatively rapid as in old people, so that in the majority of cases the affection is only recognised at the autopsy or the operating table. In characteristic cases, as in the authors', the affection begins by a phase of vague digestive troubles, perhaps subsequent to a gastro-enteritis and sometimes attributable to bad feeding. Then a dull

pain ensues, sometimes continuous, sometimes paroxysmal, rarely going through to the back; pressure in the epigastric region provokes the pain. Dyspeptic troubles consist in modifications of appetite characterised by periods of anorexia following fits of hunger, in vomiting, biliousness and constipation, more rarely diarrhœa. Acid regurgitations were well marked in one of the authors' cases. Hæmatemesis, so important from a diagnostic point of view, was wanting in two thirds of the cases terminating in death and verified by autopsy; it sometimes follows violent exertion and may be bright red or coffee-coloured. The general health is in relation to the intensity and frequency of the painful attacks. VINCENT DICKINSON.

Cultivation of the meningococcus from the eye conditions complicating epidemic cerebro-spinal meningitis (*'Montreal Med. Journ.,'* December, 1908).—McKee considers metastatic ophthalmus in epidemic cerebro-spinal meningitis not rare; many cases are mild and may be overlooked. It usually occurs in one eye only; the signs are hypopyon iritis with exudation into the papillary area, and quickly assumes the picture of pseudo-glioma. McKee describes a case in a child aged 7 years affecting the right eye, and from the anterior chamber pus was obtained from which grew the typical diplococcus; in only two previous cases was this done. The organism is easily confused with the gonococcus and the *Micrococcus catarrhalis*, and can only be distinguished by its staining reactions and cultural features. Conjunctival symptoms in epidemic cerebro-spinal meningitis may arise due to many different organisms, such as *Staphylococcus aureus*, *Bacillus influenzae*, diplobacillus, and *B. xerosis*, etc. These are easily recognised by morphology, but the Gram-negative ones, as gonococcus, *M. catarrhalis*, and meningococcus are only to be distinguished by their varied behaviour on culture mediums. J. PORTER PARKINSON.

Congenital hypertrophic stenosis of the pylorus (le rétrécissement congénital hypertrophique du pylore) (*'Gaz. des Hôp.,'* December 31, 1908, p. 1791).—Sarvonat gives a general review of the subject, chiefly characterised by an excellent bibliography. No new points are brought out. The author considers that spasm of the pylorus cannot account for all the cases. Medical treatment is recommended, at all events at first. ERNEST JONES (Toronto).

Congenital muscular atonia (l'atonie musculaire congénitale) (*'Gaz. des Hôp.,'* February 5, 1909, p. 173).—Lévi-Sirugue gives a general review of Oppenheim's disease. The article contains no new facts, but constitutes a useful summary. The cardinal symptoms of hypotonia, paresis, or paralysis, and abolition of reflexes are described in detail, and the diagnosis discussed between the condition and acute anterior poliomyelitis, progressive muscular dystrophy, Parrot's syphilitic pseudo-paralysis, and paralyzes due to various cord lesions. Spiller found changes only in the muscles, and Baudouin throughout the lower motor neuron, especially in the anterior horn cells. Thirty-three per cent. of the patients die; the sexes are equally affected. Nothing is known concerning the ætiology. ERNEST JONES (Toronto).

Two cases of Werlhof's disease in the same family (deux cas de maladie de Werlhof dans la même famille, a quinze jours d'intervalle, sous le même toit) (*'Gaz. des Hôp.,'* December 8, 1908, p. 1683).

—**Gabriel Ravarit.**—The first case was in a woman, aged 53 years, who was suddenly seized with great malaise and gastrorrhagia. On the next day diffuse purpura appeared. She died in four days. Twelve days later the granddaughter, aged 6 years, suffered similarly but to a milder degree. She recovered in ten days. The suggestion of infection is discussed.

ERNEST JONES (Toronto).

Histero-epileptic fits cured by circumcision (*'La Med. de los Niños,'* October, 1908).—**Torres and Cuadras** report the history of a child, aged 9 years, who had been always noted for violent outbursts of temper, headaches and attacks of sleepiness. The first fit occurred at the age of eight; he fell and was seized with convulsions lasting a quarter of an hour. These were repeated two or three times a day. Physical examination revealed no asymmetric or other defects; vision was normal in every respect. The later fits were preceded by auræ, headaches, optical or auditory illusions, and the convulsions involved the whole body. The child had one fit the second day after the operation, but there had been none since—which was some years ago. The cessation was not due to isolation from the family since the child was forty days in hospital before the operation, and during that time the attacks continued.

M. D. EDER.

Diabetes mellitus in a child aged 5 years, followed by pulmonary tuberculosis (*'La Med. de los Niños,'* October, 1908).—**Gonzalez** states that the child had a feeble constitution and was brought to him for increasing loss of weight with great thirst. There was considerable sugar present and the child was passing five litres of urine a day. Seven months later he found signs of tuberculosis in the lungs; the disease was very active and rapidly ended in death.

M. D. EDER.

Noma and neurosis of the upper jaw as sequel to measles; cure (*'La Med. de los Niños,'* October, 1908).—**Pares** reports this case in a boy, aged 4 years, who seemed a well-formed child. He was brought to the hospital for ulceration of the left upper jaw; there was some swelling of the retro-maxillary glands. The necrosed portions of bone were removed, the ulcer treated with the thermo-cautery, and the wound rapidly healed.

M. D. EDER.

A case of Henoch's purpura (*'St. Petersburg med. Wochens.,'* December 6, 1908).—**Zoeppfel** referred to a case in a girl, aged 11 years. The family history was *nil*. The disease commenced with diarrhœa and anorexia, followed a few days later by swelling of the ankles and purpuric eruption. Vomiting and melæna ensued with colic, and other joints were attacked. Mucous membrane of mouth was normal, but there was nose bleeding. A left-sided pleurisy followed and rise of temperature. Blood examination showed diminished reds and poikilocytosis. Recovery was complete in a few months.

M. D. EDER.

Pathology.

Myopathic rickets (*'Jahrb. f. Kinderheilk.,'* Bd. 68, Heft 6).—**Bing** reported his histological researches in twelve cases of rickets, in which four showed decided pseudo-paretic hypotonic disturbance of the muscular system with well-marked Hagenbach's symptoms; in six others there was

decided weakness and sluggishness of the muscular system, while two showed no muscular phenomena. Corresponding to the degree of the affection clinically, there was found, in some of the marked cases, a considerable organic change in the muscle; in cases less marked clinically there was found no histological change, as a rule. The author concurred with Hedinger as to the striking resemblance which the much-altered muscular tissue bore to the muscular tissue of a rhabdomyoma, and places the foregoing process in the category of a retrograde metamorphosis. He considers it "a well-marked growth disturbance of the muscular system," a dystrophy, in which the starved tissues tend to a return to the primitive tissues. In conclusion the author discussed the question of the connection of myopathic rickets and congenital myotonia. He showed diagrams in illustration of his views.

J. E. BULLOCK.

Polynucleosis in the cerebro-spinal fluid in three cases of tubercular meningitis (polynucléose rachidienne dans trois cas de méningite tuberculeuse) (*Arch. gén. de méd.*, Année LXXXVII, p. 584).—**Landowski** and **Claret** describe three such cases. They conclude that although lymphocytosis is a valuable indication of the tubercular nature of a meningitis, still the latter diagnosis should not be excluded because the cell picture is a polymorphonuclear one. It may happen that the leucocytosis in the early stage is of the polymorphonuclear variety, and later on of the lymphocytic.

ERNEST JONES (Toronto).

Anatomico-pathological and histological changes in the lymphatic glands in congenital syphilis (Alterazioni anatomico-patologiche ed istologiche dei gangli linfatici nella sifilide congenita) (*Giorn. internat. delle Scienze mediche.*, Anno XXIX, p. 481).—**Viltorio Nista**, after reviewing the subject, describes at length the findings in two cases of congenital syphilis carefully examined. He concludes that here, as in adult cases, the glands show notable lesions. These may be gummatous, inflammatory, or degenerative with sclerosis. So in the adult the glandular system may be regarded as a barrier of defence of the organism. It is probable that the malignant cases in which degenerative and amyloid changes are found diffused, scattered, but hardly any specific changes, are explicable by the fact that the mother is in an active stage of the disease, and has directly transmitted to the infant not only the virus but also quantities of toxin.

ERNEST JONES (Toronto).

Therapeutics.

Electrotherapy in paralyzes of early life (*Arch. of Pediat.*, 1908, p. 912).—**T. A. Romeiser** states that in infantile spastic paralysis not much is to be expected from electricity alone, but that this treatment is of the greatest value in flaccid paralysis. In hospital practice Romeiser uses chiefly faradic and in private practice chiefly galvanic currents. Electrical treatment must not be started until all irritative phenomena of pain and tenderness have gone, in most cases not before the end of the first month. The duration of treatment varies. Greatest improvement is during the first four to six months. Romeiser thinks that electricity is an important adjunct in the therapy of poliomyelitis though secondary to the essential treatment, which is orthopaedic and surgical in most cases.

J. D. ROLLESTON.

Fat incapacity in young infants (*'Pediatrics,'* 1908, p. 764).—**A. S. Bleyer**.—Attacks of vomiting, colic, and constipation, both in breast-fed and in hand-fed children may be due to fat incapacity. Bleyer has found that the indication for sodium citrate in such cases becomes relatively infrequent when the fats have been removed, and that a high proteid diet is then well tolerated.

J. D. ROLLESTON.

Otology, Laryngology, and Rhinology.

Suppurative middle-ear disease with mastoid symptoms and infectious pseudo-rheumatism of naso-pharyngeal origin (*'Trans. of the French Otol. Soc.,'* 1908).—**Louis Bar**, of Nice, relates the very interesting case of a girl, aged 14 years, who left Siberia with right acute middle-ear suppuration. Passing through Moscow, the sudden cessation of discharge necessitated myringotomy, after which she proceeded to Nice. Shortly before the otitis the patient had an attack of articular rheumatism, without redness, but with swelling, chiefly affecting the knee. The patient was anæmic, and had suffered for several years with foetid nasal crusts. Her nasal mucosa was sensibly atrophic and ozænatous, naso-pharyngeal mucous membrane red and inflamed, and she suffered from indigestion due to the continual swallowing of nasal mucus. When seen on January 19, 1906, she showed no trace of rheumatism. On January 24 her temperature rose to 100·4° F., the otorrhœa diminished, and pain and swelling appeared round the mastoid. She was worse next day, temperature 104° F., pulse-rate 125–140, with accentuated mastoid symptoms, and tenderness over the right iliac fossa. The question of opening the mastoid was considered, but Bar decided to wait—a delay justified by subsequent events. Next day a sudden and remarkable improvement took place, mastoid tenderness and swelling becoming markedly less and the fever ceasing. Some pupillary inequality and a little headache remained, but any other sign of meningitis was absent, and, save for the nasal condition, there was nothing to be found in the respiratory passages. On the 27th the mastoid symptoms had practically cleared up. From this time there appeared an infectious pseudo-rheumatism, with disconnected febrile attacks, manifested now in the costal cartilages, now in the vertebral spinous processes, now in the epiphyses of the long bones, now in the fingers and wrists. Usually one articulation was affected at a time, rarely several. The region was hot, red, painful, and exquisitely tender, the condition being fugitive. Finally, a systolic murmur was noted conducted towards the axilla. The patient was thus ill for two or three months, and when she left Nice she was not altogether free. Analysis of the nasal discharge showed absence of tubercle bacilli, but contained streptococci. Bar, in discussing this case, considers that the mastoid condition was independent of the middle-ear condition, and was a rheumatic inflammation of the subcutaneous cellular tissue, like that described by Trosier and Boosy, Chuffart, Brissaud, and Davaine under the title of "rheumatic œdema" and "ephemeral rheumatic nodes." He believes that the patient was chronically poisoned by her nasal condition, and, being the child of parents with a strongly marked rheumatic diathesis, the rheumatic attack was so induced. He points out also the possible connection with the "para-tuberculosis" of Poncet and Leriche, and concludes that the history of this arthritic attack is the history of a more or less latent tuberculosis.

MACLEOD YEARSLEY.

Surgery.

Observations on injuries of the neck of the femur in early life (*Med. Record*, January 2, 1909).—**Whitman** states that it is generally assumed that all fractures of the hip in early life are due to separation of the epiphysis. Thus of eighty-four cases collected from the literature by Hoffa eighty were diagnosed as epiphysal fractures, and four reported by the author as fractures of the neck. During the first decade of life at least separation of the epiphysis is uncommon, as the part is protected by a firm covering of cartilage in addition to periosteal tissue, in adolescence, on the other hand, the junction between the head and neck becoming relatively weak. The differential diagnosis between the two conditions is important as regards treatment. An injury to the hip in a healthy individual, whether a child or adolescent, which presents the physical signs of fracture is far more likely to be a fracture of the neck than a separation of the epiphysis. If, however, the patient is an adolescent of the weak, rapidly growing, or over-weighted type, if the symptoms are produced by a comparatively slight injury, and if the disability is not complete, but slowly progressive, the probability is that an epiphysal fracture has occurred. If the separation is immediate and complete the injury is generally more severe, and the disability is proportionately greater. The results of untreated cases of fracture of the neck are distortion and apparent shortening of the limb, with comparatively free movement in directions not opposed by the deformity, the limitation of movement being especially the loss of abduction due to coxa vara. In injury to the epiphysis there is, in addition, great limitation of movement, or partial ankylosis with a certain amount of shortening due to loss of growth. In a case of recent fracture of the neck traction should be applied, and the limb be fixed in a position of complete abduction in order to reduce the deformity. If union has taken place, a wedge of bone should be removed from the base of the great trochanter to restore the angle of the neck and the consequent range of abduction. In rare cases separation of the epiphysis may be complete, so that the fragments may be manipulated into place, but usually the fragments are firmly adherent and reposition is not possible without operation. In such cases the anterior surface of the joint is exposed through a straight incision in the line of the neck, and the capsule is opened. It is then often necessary to remove a thin section of bone to allow of the insertion of a chisel with which to prise the fragments apart for replacement by abduction and inward rotation of the shaft. In cases of united fracture direct operation is required for the purpose of uniting the fragments.

T. R. WHIPHAM.

Contraction of the second phalanx (*Med. Press*, March 10, 1909).—**Lotheissen** showed at the Gesellschaft der Aertze of Vienna a patient on whom he had operated for contraction of both little fingers, which became stiff without any apparent cause about the eleventh year of age. He first made an N-shaped incision, dividing the skin over the interossei and lumbricales, forming two triangular flaps. As he then found that the finger could not be straightened he resolved to divide both angles of the aponeurosis, which in this case was much wider but shorter than the normal, and thus relieved the contraction. He considered that the condition was similar to Dupuytren's contraction of the palmar fascia, which appears to originate in a chronic plastic inflammation. The patient after the operation was able

to move his fingers freely both in extension and flexion. Klein thought that many of these contractions were hereditary, like retinitis pigmentosa, which had always a family history. Tandler remarked that these cases of contraction of the little fingers were not uncommon if the statistics of the post-mortem room are to be believed. Schlesinger said that he had seen contractions in the little fingers for three generations in one family. As a rule there was an abnormal shortening of the fingers and a shrinking of the phalanges.

T. R. WHIPHAM.

Primary tumours of the adrenal gland in children (*'Amer. Journ. of the Med. Sciences,'* June, 1908, vol. cxxxv, p. 871).—**Tileston and Wolbach** give a careful and detailed description of the case of a male infant, aged 16 months, who had sarcomatous disease in the adrenal gland and cranium, with exophthalmos. The subject is generally discussed after a full review of the literature. Cases of adrenal tumours in children fall into four clinical types: (1) Those with metastases to the skull (Hutchinson's type); (2) those of simultaneous sarcoma of the liver and adrenal; (3) those associated with precocious maturity; (4) other cases. The tumours are almost invariably sarcomas. Suspicion of the malady should always be aroused in cases of orbital tumours in children; cases of chloroma can be excluded by examination of the blood, and myeloma, which is exceedingly rare in children, by examination of the urine for Bence-Jones proteid.

ERNEST JONES (Toronto).

Nerve anastomosis in infantile paralysis (*'Med. Record,'* July 11, 1908, p. 54).—**Karl Osterhaus** describes the different methods of nerve anastomosis in vogue. He reports the case of a boy, aged 10 years, who at the age of six suffered from acute poliomyelitis which left a right talipes equino-varus. A crossed transplantation between the two popliteal nerves was made, and the tendo Achillis and tibialis anticus tendon divided. The result was encouraging.

ERNEST JONES (Toronto).

Herniotomy in infants (*'Arch. of Pediat.,'* 1908, p. 925).—**A. P. C. Ashhurst** records two cases: (1) Male, aged 6 weeks, whose hernia had appeared the night before the operation; (2) male, aged 11 months, in whom a right inguinal hernia had existed since birth. Both recovered. Ashhurst has collected fifteen cases of herniotomy for strangulated inguinal hernia in infants which have been reported since Telford's paper (*v. BRITISH JOURNAL OF CHILDREN'S DISEASES*, 1907, p. 515).

J. D. ROLLESTON.

Congenital obstruction of urethra (*'Arch. of Pediat.,'* 1909, p. 58).—**T. Speese** records a case in a child born dead. Syphilis had been regarded as a possible cause of death owing to a history of repeated miscarriages. At the autopsy both kidneys were found to be enormously distended, normal tissue being completely absent. The ureters measured two centimetres each in diameter. The bladder was also distended and projected into the abdominal cavity. The cause of the obstruction was a valve-like fold situated in front of the verumontanum. The anterior urethra was normal, though owing to balanoposthitis the prepuce was œdematous.

J. D. ROLLESTON.